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AN INTRODUCTION
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PATHOLOGY AND MORBID ANATOMY.

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BY

T. HENRY GREEN, M.D., LOND.,

MEMBER OF THE ROYAL COLLEGE OF PHYSICIANS; LECTURER ON PATHOLOGY AND MORBID
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TO

WILSON FOX, M.D.,

PHYSICIAN EXTRAORDINARY TO HER MAJESTY THE QUEEN,
HOLME PROFESSOR OF CLINICAL MEDICINE, FORMERLY PROFESSOR OF PATHOLOGICAL ANATOMY
AT UNIVERSITY COLLEGE, LONDON,
AND PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL,

THIS SMALL WORK

Is Inscribed

WITH MUCH RESPECT AND GRATITUDE

BY HIS FORMER PUPIL,

THE AUTHOR.

PREFACE.

THE increasing importance which the study of Pathology and Morbid Anatomy has recently assumed in this country, has induced the author to endeavor to supply an admitted want in our medical literature, by publishing this small work on the subject. As qualifications for so doing, he claims merely to have been, during the past few years, a student and teacher in the Post-Mortem Room, and to have endeavored to make himself acquainted with the writings of others on the same subject.

This work, as its title implies, is strictly elementary. Its object is to give a brief account of the more important morbid processes which take place in the human body, in accordance with the present position of pathological knowledge. To fulfil this end, the general pathology of each process has first been described, and subsequently the same process as it occurs in the several organs and tissues of the body has been considered.

As the work is mainly intended as an elementary textbook for the student, a discussion of the different views which are held by different Pathologists respecting some of the subjects of which it treats, has been as far as possible avoided, the author having endeavored to advance those opinions which appear to him to have the greatest claim on general acceptance. For the same reason reference to the various authors has in most cases not been given, it having been deemed sufficient to append a list of the principal works which have been consulted.

Pathology has made such rapid and important advances during the last few years—mainly owing to the researches of German investigators—and the knowledge of some of the most important of the morbid processes—*e. g.*, the development of new formations, inflammation, and tuberculosis—is still so far from being complete, that the author has experienced considerable difficulty in the composition of this work. For its many imperfections he would therefore claim the reader's indulgence.

The woodcuts have, for the most part, been borrowed from other works, principally from "Rindfleisch's Lehrbuch der pathologischen Gewebelehre;" but some of them have been drawn by the artist from the author's own preparations.

In conclusion, the author must express his deep obligations to his colleague, Dr. Silver, for the trouble he has so kindly taken in helping him to revise the proof-sheets as they have passed through the press, and also for the many valuable suggestions given him in the preparation of the work.

74 WIMPOLE STREET, CAVENDISH SQUARE,
August, 1871.

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PATHOLOGY AND MORBID ANATOMY.

INTRODUCTION.

PATHOLOGY treats of the origin, nature, course, and causes of those changes in the body which constitute disease; *Morbid Anatomy*, or *Morbid Histology*, of the actual alterations in the tissues which the disease has produced. The former is therefore comparable with Physiology, the latter with the Histology of the normal tissues.

By *disease* is understood some deviation from the state of health; a deviation consisting for the most part in an alteration in the functions, properties, or structure, of some tissue or organ, owing to which its office in the economy is no longer performed in accordance with the normal standard. As *health* is itself merely a relative term and implies no definite performance of the processes of life, so *disease* is equally indefinite; it cannot be separated from health by any well-defined boundary, the one passing by insensible gradations into the other.

Disease, being thus merely an abnormal performance of those processes which constitute life, a knowledge of these must necessarily precede the study of pathology. Life comprises the formation and maintenance of the tissues and the exhibition of their various functions. Such formation and maintenance, which may be included under the general term of *Nutrition*, consist in the continuous supply of new material, the separation of this from the blood, and its appropriation by the tissues, together with the removal of the products of their waste. *Function* is the special manifestation of the life of the part, as distinct from its growth and the maintenance of its structure; in the secreting cell, consisting in the alteration of the substances abstracted from the blood to fulfil some special purpose in the economy; in nerve, in the transmission of impulses of motion and sensation, &c. The performance of function is obviously dependent upon the state of nutrition. When both of these are normal the condition is one of *health*, when abnormal one of *disease.*

As in health the nutrition is principally dependent upon the tissues themselves, these abstracting, appropriating, and altering the material which is supplied to them by the blood; so in disease it is the *tissues* which play the most important part, and alterations in them are amongst the most frequent of the morbid processes.

The supply and composition of the *blood* must at the same time constitute a most important cause of abnormal nutrition. In regarding the blood, however, as a cause of disease, it must be borne in mind that this fluid is in a state of constant dependence upon other parts; its component elements are derived from external sources and undergo continual change, and although possibly under certain circumstances it may become altered by virtue of changes in the nutritive activity of its corpuscular elements, alterations in its constitution must in most cases result either from some change in the process of its formation, as from the ingestion of improper or insufficient food, mal-assimilation, or disease of the lymphatic structures; from changes in the secretory or excretory processes; or from the introduction of foreign substances, derived from extraneous sources. Whilst, therefore, alterations in the composition of the blood may be important agents in the production of abnormal nutritive changes, they almost invariably depend upon some antecedent condition, and can rarely be regarded as the *primary* cause of disease.

The blood may also become a cause of disease, owing to an abnormal activity of its white corpuscles and their migration through the walls of the bloodvessels into the surrounding tissues, where they may constitute centres of morbid nutritive changes. (See "Inflammation.")

Lastly, the influence of the *nervous system* must be taken into account in considering alterations in nutrition and function. This not only influences the circulation and supply of blood, but also tissue-change; recent researches tending to show that nerve-fibres terminate in the ultimate elements of nearly all tissues.

The *first* part of this work will be devoted exclusively to the consideration of morbid processes which are characterized mainly by alterations in nutrition; the *second*, to those in which an altered nutrition is associated with changes in the blood and circulation, constituting "inflammation;" and the *third*, to changes in the blood and circulation alone.

CHAPTER I.

THE "CELL."

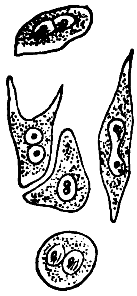
As the most important element in nutrition, both in health and disease, is the activity of the tissues themselves—the supply of nutritive material, although an essential, being merely a *passive* part of the process—it becomes necessary to consider, somewhat minutely, those parts of the tissues in which this activity resides.

Ever since Schwann discovered the cellular nature of animals, and established the analogy between animal and vegetable cells, there has been a gradually increasing conviction amongst physiologists, which has now become a universally accepted physiological and pathological doctrine, that the *cell* is the seat of nutrition and function; and further, that *each individual cell* is itself an independent organism, endowed with all those properties, and capable of exhibiting all those active changes which are characteristic of life. Every organized part of the body is either itself a cell or is derived from cells, and the cells themselves originate from pre-existing cells, and under no circumstances do they originate *de novo.*

Whilst therefore the whole body is made up of cells, or of substances derived from cells, and the cell is itself the ultimate morphological element which is capable of exhibiting any manifestation of life, it must be borne in mind, that in a complex organism, the phenomena of life are the result of the continued activity of innumerable cells, many of which possess distinct and peculiar functions, and which by their combination become endowed with new powers, and exhibit new forces; so that, although each individual unit possesses an independent activity, it is in a state of constant dependence upon others with which it is more or less intimately associated.

CONSTITUTION OF CELLS.—When the analogy was established between the animal and vegetable cell, the former was held to be constructed in all cases upon the same principle as the latter, and to consist of a *cell-wall*, inclosing a cavity in which were contained a *nucleus* and *fluid contents*. (Fig. 1.) This was the idea of the cell held by Schwann and Remak, and supported especially by Virchow, who maintained that these three constituents were essential to its vitality and existence.

FIG. 1.



Cells from a Cancer. Showing cell-wall, cell-contents, nuclei, and nucleoli. The nuclei dividing.

During recent years, however, this definition has been modified. The existence of a cell-wall was in many cases not evident: in the embryonic cells, in those of many rapidly growing new formations, and in the corpuscles of blood, pus, and mucus, no limiting membrane could be demonstrated. This led to a new definition of the cell by Leydig and Max Schultze, who held that a little mass of matter inclosing a nucleus was all that was necessary for its constitution. The latter of these physiologists not only rejected the cell-wall as an essential constituent, but established the identity of the mass of matter (cell-contents) with animal sarcode — a contractile substance existing in the lower animals—and showed, that like it, it was endowed with the power of spontaneous movement: this substance he called *protoplasm*. He further pointed out, that the existence of a distinct cell-wall was the result of a retrograde process taking place in the outer layers of the protoplasm, and that the latter was the real seat of the activity of the cell. These views closely correspond with those held by Dr. Beale in this country.[1]

The definition of a cell has been still further modified by Brücke, Stricker, and others, who consider that the existence of a nucleus is not essential to its constitution. This opinion is principally based upon the fact, that in the cryptogamia, and in some of the lowest animal forms, cells occur in which no nucleus is visible.

It would thus appear probable that a simple mass of protoplasm may, in some exceptional cases, be all that is necessary to constitute a cell—*i. e.*, an elementary organism, capable of exhibiting independently all the phenomena of life; but that the nucleus is an exceedingly constant, and almost invariable, constituent. The cell-wall is much less constant, and being the result of a retrograde change in the outer layers of the protoplasm, it must be regarded, in point of vitality, as inferior to the rest of the cell.

---

[1] Dr. Beale calls the protoplasm *germinal matter* or *bioplasm*; the cell-wall, *formed material*.

Protoplasm itself is a homogeneous structureless material, although as it is met with in cells it usually contains adventitious matters—as granules of fat, pigment, &c.—which are either the result of its metamorphosis, or have been taken up from without. In consistence it is subject to constant variations, being sometimes perfectly fluid, at others more or less solid and gelatinous. The cell-wall, when it exists, is of much firmer consistence than the protoplasm. In some cells the protoplasm constitutes but a small proportion of the cell-contents, other substances peculiar to the cell being associated with it; as, for example, fat in the cells of adipose tissue. (See also fatty infiltration of liver cells, Fig. 10.)

The nucleus is much more constant both in size and form than the cell. It is usually spherical or oval in shape, and often contains one or more minute, round or angular bodies, termed *nucleoli.* It offers a greater resistance to chemical reagents than the other constituents of the cell, and in disease often remains after these have been destroyed. It is also stained more deeply by carmine. Structurally, it appears often to be perfectly homogeneous, or faintly granular; in some cases it is invested by a limiting membrane, and is thus of a vesicular nature. Several nuclei may be contained within the same cell.

PHYSIOLOGY OF CELLS.—The cell being, as already stated, the seat of all those nutritive and functional processes which are characteristic of life, the question arises, as to what part is played by its respective constituents, and whether the cell-wall, the protoplasm (cell-contents), and the nucleus have different offices.

The cell-wall being the result of a retrogressive change in the protoplasm, it cannot be regarded as taking any part in the life of the cell, the activity of which is diminished by its existence, as is also its power of reproducing itself by division. It is in old cells that a cell-wall is most frequently met with; in those newly formed it is usually entirely wanting.

The nucleus has always been looked upon as the seat of the nutrition, as distinct from the specific functions of cells, and has been supposed to play an important part in their multiplication and reproduction. The fact that when a cell divides, the division usually commences in the nucleus, and only subsequently takes place in the rest of the cell, would appear to favor this view; as would also the great uniformity of the nucleus both in size and form, whatever be the functional nature of the cell. It must be borne in mind, however, that non-nucleated cells may multiply, and that nucleated cells have been observed to divide, the nucleus itself taking no part in the process.

Whatever be the part played by the nucleus, there can be no doubt that the protoplasm is the most important factor of the cell, and it may itself be the only constituent. The spontaneous movements, alterations in form, and migratory powers characteristic of young cells, are due to the protoplasm; such movements are observed in the cells of the embryo, in lymphatic and young epithelial cells, in some of the cells of connective tissue, and in white blood-corpuscles and pus-corpuscles.

The protoplasm, as already stated, may be the sole seat of the nutritive and formative power of the cell; it would appear, however, probable that it is especially concerned in the performance of function, and that the specific functional peculiarities of cells are dependent rather upon it than upon their other constituents. The volume and consistence of the protoplasm varies in different cells, and in the same cell at different times, and under different circumstances. It is apparently capable of imbibing and giving up fluids, at the same time undergoing corresponding alterations in volume. These considerations render it probable that it is the seat of the selective power of the cell, and of those other properties which represent its specific functions.

GENESIS OF CELLS.—The proposition of Virchow, that every cell originates directly from a pre-existing cell, forms the basis of the pathology of the present day. To Remak, however, must be ascribed the merit of having first established the cellular origin of the tissues.

The multiplication of cells may take place in three ways,—by *simple division*, by *gemmation*, and by *endogenous growth*. In the first two methods the cell breaks up into fragments; in the last new cells originate within the parent cell. The process is obviously associated with growth and increase of the protoplasm.

The multiplication by simple division is the most frequent method. The cell divides and forms two cells, and each of these again divides and forms two more, and so on. In nucleated cells the nucleus as a rule divides first. The nucleus, however, may divide and multiply within the cell without any division of the cell taking place. The existence of a dense cell-wall interferes with multiplication by simple division.

In multiplication by gemmation, a small portion of the protoplasm projects from the cell, and becomes detached by constriction at its base, and thus forms a new cell. This is much less frequent than the former process.

Endogenous multiplication occurs principally in cells possessing a dense cell-wall, as in some varieties of epithelium. The protoplasm divides within its limiting membrane, and thus a number of new cells are formed within the parent cell, from which they are subsequently liberated either by the destruction of its wall, or by virtue of their own amœboid activity.

CHAPTER II.

NUTRITION ARRESTED.

The absolute and permanent arrest of nutrition constitutes local or systemic death. Under this head are included the three following processes:

1. *General or Systemic Death.*—In this, the arrest is general; the nutritive processes cease throughout the whole body.
2. *Gangrene or Necrosis.*—The arrest is local, and confined to a particular part, which, when dead, retains to a greater or less extent its external form and anatomical characters.
3. *Molecular Death or Necrobiosis.*—The arrest is also local, but usually results from a previous gradual impairment of the nutritive processes, which ultimately entirely cease in some of the histological elements. The dead part is a granular débris in which all trace of the original structure is lost. This will be considered under "Atrophy and Degeneration."

GANGRENE OR NECROSIS.

Gangrene or Necrosis is the complete and permanent arrest of nutrition in a part—occurring for the most part more or less suddenly—which, when dead, retains to a greater or less extent its external form and anatomical structure. The series of processes by which this is brought about is called *Mortification*, the dead tissue, a *Sphacelus* or *Slough*. In bone the process is called *Necrosis*, the result, a *Sequestrum*.

The arrest of nutrition is followed by the complete cessation of all the evidences of life both functional and physical; and the part thus removed from the influence of the "vital forces" undergoes those chemical and physical changes which are common to inanimate organic matter. In a limb, for example, there is paralysis of motion and sensation, coldness, dryness of the surface; the natural firmness and elasticity are lost; and it becomes soft and doughy, the coloring matter escapes from the blood-corpuscles, and dissolved in the liquor sanguinis permeates and stains the tissues. Evaporation from the epidermis is hindered: consequently, if the limb contains much blood, the transuded serum and the watery constituents of the tissues form large bullæ on the surface. As decomposition proceeds, gases are generated in the part;—principally sulphuretted hydrogen, ammonia, nitrogen, and carbonic acid: these give rise to the emphysematous crackling which is so often associated with the gangrenous process. The tissues at the same time undergo a process of softening or liquefaction, the limb becomes exceedingly offensive, and changes from a reddish color to a brownish or greenish-black, this is owing to changes in the transuded hæmatine. If the limb does not contain a large amount of blood, and evaporation is allowed to go on freely from the surface by the destruction of the epidermis and rupture of the bullæ, it may dry up—the process of decomposition gradually ceasing—and be converted into a black shrunken mass, which undergoes but little further change: this constitutes *Dry Gangrene* or *Mummification*. If, on the other hand, as is more commonly the case, the gangrene is associated with venous obstruction, and thus the return of blood and absorption of fluids are prevented, the evaporation from the surface is rarely sufficient to dry the limb, and consequently, the process of decomposition proceeds until it is completely disorganized: this is *Moist Gangrene*.

The characters of the dead part vary with its vascularity, its structure, the cause of the gangrene, the acuteness of the process, and the possibility of the access of atmospheric air. The more vascular the tissue, the softer its structure, and the more it is exposed to the atmosphere, the more rapidly and completely does it undergo decomposition. Bone, cartilage, and tendons, which are firm hard tissues, containing comparatively but few vessels, undergo very little alteration in structure and form; whereas the softer parts are much more rapidly and completely destroyed.

The occurrence of decomposition manifests itself in the first place in the blood contained in the part: this fluid undergoes the earliest and most rapid change. The hæmatine escapes from the red corpuscles, partly by exudation, and partly by the destruction of the corpuscles themselves, and dissolved in the liquor sanguinis permeates the surrounding tissues. The corpuscles are ultimately completely annihilated, nothing remaining but a few minute granules.

The staining of the tissues with hæmatine, commonly known as *post-mortem staining*, is very characteristic. All the tissues are more or less affected, the lining membrane of the bloodvessels, which is in immediate contact with the blood, being naturally more so than other parts. The staining is of a uniform pinkish-red color, thus differing from the punctiform and striform redness of hyperæmia, from which it must be carefully distinguished. The amount of staining is in proportion to the rapidity with which decomposition has taken place, and to the amount of blood contained in the part at the time of death. It is usually most marked in the lining membrane of the heart and large bloodvessels.

Rigor Mortis.—In muscle the arrest of nutrition is accompanied by a state of rigidity, known as the *Rigor Mortis.* This is a peculiar condition of the muscles observed in almost all bodies after death, in which they become firm and somewhat shortened, as though in a state of chronic contraction. It comes on as soon as the muscles have lost their irritability, *i. e.*, their capability of responding to artificial stimulation; in other words, as soon as the nutritive processes have completely ceased. The time of its appearance will therefore depend upon the state of nutrition of the muscles at the time of death; the more healthy and vigorous this is, the longer it is before it completely ceases, and consequently the longer it is before the rigor mortis supervenes. The length of its duration and its intensity are in direct proportion to the lateness of its appearance. In people, for example, who are in perfect health, and die suddenly, as from accident, the rigor mortis does not usually come on until from ten to twenty-four hours after death, it is very marked, and often lasts two or three days. In those, on the other hand, who die from some exhausting disease, as phthisis or the adynamic fevers, in which the nutrition of the muscles becomes much impaired, the rigor mortis appears very soon sometimes only ten minutes after death: it is very slight and may pass off in less than an hour. It has been said that in case of death from poisoning by carbonic acid and sulphuretted hydrogen, from lightning, and from some of the severer forms of the adynamic fevers, the rigor mortis is entirely absent. It is doubtful, however, if this is the case, as the rigor mortis has probably escaped observation, owing to its early supervention and rapid disappearance. As soon as the rigor mortis has passed off, decomposition of the muscular tissue commences.

The rigor mortis occurs not only as the result of systemic and local death, but it may also be induced artificially by temporarily arresting the nutrition of the muscle. If the supply of blood to a muscle is cut off by the application of a ligature to the artery supplying it, it quickly passes into a condition which is indistinguishable from the rigor mortis; if the ligature be removed sufficiently early this disappears, and the vitality of the muscle is restored.

With regard to the nature of the change, it was formerly supposed to be a spontaneous contraction, the last act of vitality on the part of the muscle. More recently, however, Kühne and others have shown that it is really owing to the presence of a firm albuminoid compound (Myosin), which is separated from the fluid of the muscle when its nutrition has ceased, and coagulates in its substance, thus causing the firmness, hardness, and opacity, which disappear as soon as its disintegration and decomposition commence. The transverse striation of the fibres then becomes indistinct, and gives place to irregular rows of granules and fat molecules, the muscle softens, its sarcolemma is destroyed, and ultimately nothing remains but a soft structureless débris.

A similar coagulation appears to take place in the cells of plain involuntary muscle, and here also a rigor mortis occurs. In the cells of other tissues—as the Malpighian layer of the skin, the connective tissues, and glandular organs—a coagulation of the protoplasm has been observed on the cessation of the nutritive processes; the cells become cloudy and granular, and then break up into molecules of various sizes.

The termination of the gangrenous process varies; it may, after involving a greater or less extent of tissue, become arrested, and a "line of demarcation" form between the dead and living parts (*Circumscribed Gangrene*), or the process may continue to extend without any such attempt at recovery (*Diffuse Gangrene*).

The dead tissue, the sphacelus or slough, acts as a foreign body, and as such sets up inflammatory changes in the adjacent structures; and it is by this means that it is ultimately removed, or becomes encapsuled. The tissues immediately surrounding the necrosed part are thus in a state of inflammation, as is evidenced in external structures by their swelled condition, red color, and high temperature. As the gangrenous process ceases, the necrosed fragment becomes limited by this line of inflamed tissue, which constitutes the "*line of demarcation*" between the dead and living parts. Along this line a process of ulceration and suppuration takes place, and by means of this the dead mass is gradually separated from the surrounding structures. The ultimate termination of the process depends principally upon the situation of the affected part; if this is superficial the slough is thrown off as in external parts, the intestines, pharynx, &c., an ulcerated surface being left. If the dead mass is deeply seated, its removal becomes possible only by the extension of the necrotizing process to the surface, as is exemplified by the spontaneous removal of necrosed bone through fistulous openings in the soft parts, and by the opening of an abscess. In other cases the inflammatory process which takes place in the tissues surrounding the dead part is less intense, and the formation of pus is less abundant, and is soon followed by that of connective tissue, a layer of which is ultimately formed around the necrosed mass by which it becomes *encapsuled.* This occurs especially in internal parts; examples of it are furnished by foreign bodies, masses of tubercle, hemorrhagic infarcts, accumulated epithelial products, portions of necrosed bone, and a fœtus in the abdominal cavity, all of which may thus become surrounded by a layer of connective tissue. The part when thus encapsuled is rendered inert, and no longer acts as an irritant to the tissues in which it lies; it undergoes a gradual process of absorption and drying up, and often becomes calcified.

Causes.—The causes of gangrene may be divided into those which interfere with the supply of nutritive material, and those which directly destroy the vitality of the histological elements, so that they are no longer able to perform their functions.

A. The supply of nutritive material may be interfered with by—

1. *Obstruction in the Arteries.*—This is a common cause of gangrene. The obstruction may be caused by a ligature, by compression of the vessel, by solution of its continuity, by thrombus

or embolism, and by disease of the arterial coats. If the obstruction is complete and a collateral circulation cannot be established, death of the part quickly ensues. Obstruction to the arteries alone, the return of blood by the veins not being interfered with, usually produces the dry form of gangrene.

2. *Obstruction in the Capillaries.*—Obstruction here is usually the result of pressure upon and stretching of the vessels. This may take place by the accumulation of inflammatory products, new formations, hemorrhage, &c., all of which by the pressure they exercise upon the capillaries may cause gangrene. The opening of an abscess takes place partly in this way,—the pressure exercised by the pus upon the tissues between it and the surface arrests the circulation, thus causing the death of these tissues, and so allowing the pus gradually to approach nearer and nearer the surface, until it ultimately escapes. Perforation of the pleura in abscess of the lung is another example of the same process. The necrosis of the superficial layers of bone which so frequently results from periostitis, is in the same manner caused by the inflammatory products compressing the nutrient vessels of the bone. New formations and tumors may also, by interfering with the circulation in neighboring structures, cause their death, as is exemplified by the molecular death which occurs in cirrhosis.

3. *Obstruction in the Veins.*—Obstruction to the return of blood by the veins must be so complete in order to arrest nutrition, that it is in itself rarely a cause of gangrene. It is when associated with obstruction in the arteries, that it constitutes an important agent in producing this result. This combination of venous and arterial obstruction is seen in a strangulated hernia, in the invagination of a portion of the intestine, in the constriction of a part by a tight bandage, and in contusions and lacerations of the soft parts in which both arteries and veins are injured, or become strangulated by the tension of the tissues which subsequently ensues. In all cases in which gangrene is associated with venous obstruction it is of the *moist* variety.

4. *Diminished Cardiac Power.*—This, like venous obstruction, is seldom independently a cause of gangrene. In cases, however, of excessive general debility, or disease of the cardiac substance, the consequent diminution in the contractile power of the organ, materially aids the foregoing causes in producing a fatal blood-

stasis. The arrest of the circulation in "Senile Gangrene," and the sloughing of the back which so often occurs in adynamic fevers, and chronic exhausting diseases, are in great measure the results of diminished cardiac power.

B. Destruction of the vitality of the histological elements may be caused by—

1. *Inflammation.*—The effect of the inflammatory process is to impair the vitality of the affected part; and the intensity of the process may be so great as to completely destroy vitality and cause gangrene. This result is undoubtedly aided by the accompanying tension and blood-stasis. Gangrene from inflammation has been called *inflammatory*, *acute*, or *hot* gangrene. It is always of the moist variety, and is characterized by the heat, swelling, tension, and redness of the affected part, which quickly becomes flaccid, cold, of a greenish-black color, and exhales an offensive odor. Certain forms of inflammation have a special tendency to terminate in gangrene; this appears to depend upon their specific characters. Diphtheria, erysipelas, carbuncle, and "hospital gangrene," are of this class. In all cases the more impaired the nutrition of the part which becomes the seat of an inflammatory process, the more likely is this to cause its death.

2. *Mechanical Agencies.*—Under this head are included external violence, heat, cold, and corrosive substances, all of which, by directly and completely destroying vitality, may be causes of gangrene. In most cases, however, this result is preceded by more or less inflammation.

3. *Poisons introduced into the Circulation.*—Phosphorus, ergot of rye, farcy, and glanders are the most important of these. The necrosis of the jaw which results from phosphorus appears, however, to be owing to the direct contact of the phosphorus with the bone. The gangrene of the extremities which sometimes follows the long-continued ingestion of ergot, is probably owing to that contraction of the small arteries which this substance produces.

SENILE GANGRENE.—This is a form of mortification which affects especially the lower extremities of old people, and is the result of several of those conditions which have already been enumerated as causes of gangrene.

The primary change usually takes place in the arteries of the limb. These become the seat of atheroma or calcification, in consequence of which the circulation is interfered with, and the vitality impaired. This is evidenced by coldness of the feet, cramps, and other abnormal sensations, which are usually experienced by the patient some time before the gangrene sets in. This tendency is materially increased by simultaneous atrophy, or degeneration of the muscular substance of the heart itself. The combined effect of the diminished *vis à tergo* and arterial degeneration may, in some cases, be alone sufficient to cause arrest of the circulation and thrombus in the vessels of the limb, and thus to cause gangrene: much more commonly, however, there is some determining cause, as a slight abrasion of the foot, a bruise, injury to a corn, or excess of heat or cold, which sets up inflammation in the already weakened part, and thus by still further impairing its vitality, and obstructing the circulation in it, causes its death. Senile gangrene is usually of the *dry* variety.

CHAPTER III.

NUTRITION IMPAIRED.

It has been seen in the preceding chapter that the absolute arrest of nutrition is followed by the complete cessation of all manifestations of vitality and function, constituting local or systemic death. Those conditions must now be considered in which the interference with nutrition, for the most part, falls short of absolute arrest, and in which, although vitality is impaired, death is only an occasional sequence. Such conditions are comprised under " Atrophy," and " Degeneration."

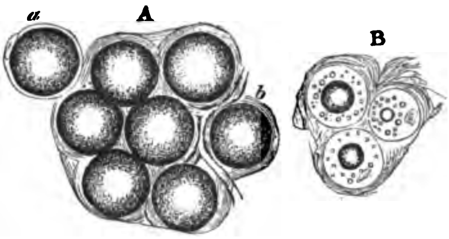
ATROPHY.

Atrophy is a diminution in the amount of a tissue, owing either to diminution in the *size*, or diminution in the *number*, of the histological elements of which it is composed. It is attended by loss of weight, and impairment of function.

When the elements are diminished in *size* only, it is called *Simple Atrophy;* when the *number* is diminished, it is called *Numerical Atrophy.* These two varieties are often associated, being different stages of the same process: *simple* atrophy may, however, exist without *numerical* atrophy, but *numerical* never exists without *simple.*

I. SIMPLE ATROPHY.—Simple diminution in the size of the elements of a tissue is by far the most common condition met with in atrophy. It is well exemplified by what takes place in ordinary emaciation, in which the fat gradually disappears from the subcutaneous adipose tissue. Adipose tissue is merely a connective tissue, the cells of which are distended with fat. When a person emaciates, the fat is gradually removed from the cells, so that they diminish in size, and the fat which completely filled the cell may be reduced to a few isolated drops: the cell-wall and nucleus at the same time often become distinctly visible (Fig. 2). Here there is no destruction of the cells, no diminution in their number, but simply the removal of some of their contents.

FIG. 2.



Adipose Tissue. A. Normal. B. Atrophic, from a case of phthisis. *a*. A single fat-cell with cell-wall, nucleus, and drop of fat. x 300. (Virchow.)

This diminution in the size of the elements may take place in any tissue. The cells of all glandular organs may thus become atrophied, and so produce a diminution in the size of the whole organ: this is constantly met with in the liver, kidneys, mammary gland, spleen, testicles, lymphatic glands, and other parts. Muscular tissue in the same way atrophies by the diminution in the size of its primitive fasciculi: this is seen in the heart and in the voluntary muscles.

In all these cases the elements remain almost unchanged; and hence all that is necessary for the restitution of the tissue is an increase in their nutritive activity, and the assimilation of more material.

II. NUMERICAL ATROPHY.—This is an advanced stage of the former process: the elements are not only diminished in size, but some of them have actually perished and ceased to exist as vital agents.

This destruction of histological elements which occurs both in atrophy and in degeneration—and is called by Virchow, *Necrobiosis*—must be distinguished from the death of circumscribed portions of tissue which constitutes gangrene and necrosis. The two processes resemble one another in so far as death is common to both of them. In *Necrobiosis*, however, the change is a molecular one; there is a gradual exhaustion of vital power, a molecular disintegration and destruction of elements, so that at the termination of the process all that remains is a granular débris, in which no trace of the former structure of the part can be discovered. The death and desquamation of the superficial layers of the epidermis is a well-known example of this molecular change. *Gangrene*, on the other hand, affects circumscribed tracts of tissue, and is the result of some sudden arrest of nutrition, as distinguished from the gradual exhaustion of vital power. Death is more suddenly induced, and a necrosed mass remains at the termination of the process in such a condition that the structure of the part can usually be recognized.

Numerical atrophy is thus of much graver import than that in which the elements continue to exist as such. In it, restitution is only possible by the production of new elements, whereas in simple atrophy, repair can be effected without new formation.

Atrophy may be *general*—affecting to a greater or less extent all the organs and tissues of the body, or it may be *partial* and limited to particular parts. General atrophy is usually *simple*, and is rarely accompanied by destruction of elements. It affects in the first place the subcutaneous adipose tissue, then the adipose tissue in other situations, as that surrounding the viscera and in the omentum, then the muscles and glandular organs, and lastly the nervous and osseous structures.

Although " atrophy " in its strict signification consists simply in a diminution in the size or number of the component elements of a tissue, it is rarely a perfectly simple process, but is usually associated with more or less *fatty degeneration*. This is owing to the fact that whenever the nutrition of a part is so much interfered with as to cause it to atrophy, it is very prone to undergo fatty changes; and it will be seen when speaking of " fatty degeneration," that this process owes its origin to the same causes as atrophy itself.

CAUSES.—In speaking of the causes of atrophy, it will be necessary to distinguish between those which act upon the tissues generally, and those which have merely a local influence.

General Atrophy may be caused by—

1. *Deficient Supply of Nutritive Material.*—Whatever interferes with the supply of nutritive material to the tissues will be followed by their atrophy. Deficient supply of food; obstruction to the passage of the food into the stomach or intestines, as in stricture of the œsophagus or pylorus; the mal-assimilation which results from the various conditions giving rise to dyspepsia; interference with the absorption of the chyle, from obstruc-

tion of the thoracic duct, or disease of the mesenteric glands constituting the so-called "tabes mesenterica;" may all in this manner be causes of general atrophy.

2. *Excessive Waste.*—All those conditions which are attended by the loss of large quantities of nutritive material, may be causes of general atrophy. Such conditions are furnished by continuous hemorrhages, profuse and long-continued suppuration such as occurs in caries and empyema, diarrhœa, and the excretion of large quantities of albumen or sugar as in Bright's disease or diabetes. The waste resulting from the increased tissue-change which accompanies acute febrile diseases, must also be included under this head.

3. *Impaired Nutritive Activity.*—This constitutes an important element in the production of the atrophy of old age,—*senile atrophy.* As life advances, the vitality of the elements gradually diminishes, their ability to separate nutritive material from the blood and to assimilate it for their own maintenance becomes less and less, and hence they gradually atrophy, and ultimately all manifestations of their vitality cease.

Although general atrophy may thus be referred to one of the foregoing causes, it is rarely a simple process, but usually depends upon the combined influence of two or more of them. The atrophy associated with pulmonary phthisis, for example, results partly from the loss of nutritive material in the profuse expectoration and diarrhœa, partly from the deficient supply consequent upon the interference with assimilation by the structural changes in the stomach and intestines, which accompany it, and partly from the increased tissue-change In senile atrophy, again, in addition to the general diminution of nutritive activity, there is frequently some condition of the digestive organs interfering with assimilation, which materially aids in producing the ultimate result. The atrophy which accompanies the acute febrile diseases is by no means a simple process, increased tissue-change, loss of appetite, and interference with assimilation being all essential parts of it.

Partial Atrophy may be caused by—

1. *Imperfect Supply of Blood.*—The effect of interfering with the supply of blood to a part will depend upon the extent of the interference; if it is entirely cut off the part will die (see "Gangrene"), if merely diminished it will atrophy.

Diminished supply of arterial blood is a common cause of atrophy, and may be brought about in various ways. The nutrient vessels may be obstructed by pressure exercised upon them, within or without the organ to which they are distributed. In cirrhosis of the liver, the increased growth of interlobular tissue by its pressure upon the capillaries causes atrophy of the secreting structures. In other cases the supply of blood is diminished by interference with the circulation at some distance from the part, as by the pressure of a tumor upon the artery leading to it. The atrophy of the proximal end of the shaft of a bone, after fracture above the point of entrance of its nutrient artery, is due to the same cause.

The atrophy which results from pressure exercised directly upon the part itself, is probably also partly owing to the consequent interference with its supply of blood. Atrophy of the sternum from the pressure of an aneurism, atrophy of the kidney from the pressure of retained secretion—as in enlarged prostate, &c., and atrophy of the skull in chronic hydrocephalus, are well-known examples of these atrophies from pressure.

Mechanical congestion in the same way is not an uncommon cause of atrophy; the circulation is impeded, the blood is not returned normally by the veins, hence there is deficient arterial supply, and atrophy results.

2. *Diminished Functional Activity.*—This is the most common cause of atrophy, many examples of which are furnished both by physiological and pathological processes. After birth those parts which are no longer required to serve any purpose in the economy gradually atrophy and waste; the ductus arteriosus, the umbilical arteries and vein, the Wolffian bodies, and later, the thymus gland, all in this manner disappear. The involution of the uterus after delivery, the wasting of the spleen and lymphatic glands in advanced life, and of the lower jaw after the loss of the teeth, are other physiological examples of atrophy from this cause.

Muscles which from any cause have long remained inactive, atrophy. This is seen in the various forms of paralysis, especially in the so-called "essential paralysis" of children; also, in limbs which have become incapacitated, either on account of anchylosis, or of chronic diseases of the bones or joints.

After the establishment of an artificial anus, the lower part of the intestine atrophies, and becomes converted into a fibro-cellular cord.

Bones in the same manner atrophy for want of use. After the amputation of a limb the cut end of the bone atrophies; and atrophy of the orbit follows extirpation of the eyeball.

Interference with the function of nerves is also followed by their atrophy; this is seen in the atrophy of the optic nerve in some cases of blindness.

3. *Increased Functional Activity.*—This may occasionally be a cause of atrophy; much more commonly, however, it is a cause of hypertrophy. Some glands atrophy from excessive use, especially the testicle. The brain may also atrophy from overwork.

4. *Inflammation.*—This is a common cause of atrophy; the vitality of the tissue becomes impaired by the inflammatory process, and it consequently atrophies, degenerates, or dies. This will be considered when treating of "Inflammation."

5. *The Action of Special Substances.*—Certain substances administered internally appear to be capable of producing atrophy: iodine, bromine, mercury, lead, and the alkalies may be enumerated amongst the most important of these. Iodine and mercury exercise an influence upon the lymphatic system, and bromine upon the organs of generation.

6. *Nervous Influence.*—Respecting the influence of the nervous system as a direct cause of atrophy, little is certainly known. That atrophy is a frequent sequence of changes in the nervous centres, there can be no doubt; but it is probably in most cases to be attributed to an *indirect* influence. The atrophy of muscles, for example, which have become paralyzed from lesions in the brain or spinal cord, is rather the result of the consequent interference with their function, than of any direct influence upon their nutrition. In some cases, also, changes in the nervous system may cause atrophy by affecting the size of the blood-vessels, and so interfering with the supply of blood. As, however, nutrition appears to be more or less under the influence of the nervous system, it is probable that nervous influence is sometimes a *direct* cause of atrophy.

PHYSICAL CHARACTERS.—The estimation of atrophy is often a matter of considerable difficulty: the great criterion is, diminution in absolute weight. The weight of an organ, however, varies considerably in health: it varies with the weight of the body as a whole, and it may be less than natural from incomplete development. The same is true also of the muscular and osseous systems. An accumulation of blood and serosity in an organ may again increase its weight, and thus constitute a source of fallacy: this is often the case in organs which have been for some time mechanically congested, in which, although their size and weight may be increased, their tissue is considerably diminished in amount.

Organs which are atrophied are diminished not only in weight, but usually also in size. In most cases they contain less blood, they are drier, firmer, and more fibrous in consistence than in health. Their functional powers are invariably diminished.

The whole of the textures of which an organ is composed may suffer; some, however, do so more than others. The fibrous constituents, instead of diminishing, may rather increase in amount. This is especially the case in senile atrophy: hence the firmness, toughness, and loss of elasticity so commonly met with in the atrophied parts. In glandular organs, the secreting cells are usually the first to show signs of atrophy: they become smaller, and are often finely granular, from the presence of molecular fat: the vessels and nerves also share in the wasting process. In the subcutaneous cellular tissue the fat is gradually removed from the cells, adipose tissue becoming common connective tissue. In muscles the primitive fasciculi become smaller, and their transverse striæ gradually disappear; ultimately the whole of the contents of the sarcolemma may be entirely removed, and nothing remain but fibrous tissue: this process is usually accompanied by more or less fatty degeneration.

Atrophy of bone is always attended by a diminution in weight, but not always by a diminution in size. The compact and cancellous tissue may gradually become absorbed, and the medullary canal diminish in size, the whole bone thus becoming smaller; this has been called *concentric atrophy*, and is met with especially in the long bones, in cases of long-standing anchylosis, dislocations, or paralysis. In other cases there is no diminution in the size of the bone, but merely a gradual conversion of compact into cancellous tissue: this, in contradistinction to the former variety, has been called *eccentric atrophy*, and is usually met with as a senile change.

DEGENERATION.

The "Degenerations" include a class of morbid processes which are characterized by an alteration in the *quality* of the tissues, and which, like atrophy, are attended by impairment of function, and often by annihilation of histological elements.

The alteration in the quality of the tissue results either from its direct metamorphosis into a new material, or from its infiltration with some substance which has been conveyed to it from without.

Atrophy and degeneration thus so far resemble one another, that in both processes nutrition is impaired and function interfered with. In atrophy, however, nutrition is simply altered in *quantity*, the waste of the tissue is in excess of the assimilation of new material, and, consequently, there is a diminution in the amount of the tissue, and an impairment of its functional powers. In degeneration, on the other hand, nutrition is altered in *quality*, a new substance exists in the tissues, which either originates in the tissue itself, or infiltrates it from without: this is attended by impairment of the vitality and functions of the elements of which the tissue is composed, resulting either from the presence of the new material, or dependent upon the same conditions as those which give rise to its formation.

Causes.—Of the causes of the Degenerations as a class, but little can be said, the various forms depending for the most part upon different conditions. These will be described under their respective heads. Fatty and calcareous degeneration have, however, many points in common, and they are both intimately connected with the atrophic changes; atrophy, fatty, and calcareous degeneration are indeed frequently different stages of the same process, and the causes of these two forms of degeneration will thus be seen to be very similar to those already enumerated as causes of atrophy.

The Degenerations may be divided into two classes—the *Metamorphoses* and the *Infiltrations.*

1. The Metamorphoses.—These are characterized by the direct metamorphosis of the albuminoid constituents of the tissues into a new material. This is usually followed by the destruction of the histological elements and the softening of the intercellular substance, so that ultimately all trace of structure may be lost,

and function be completely arrested. The Metamorphoses include Fatty, Mucoid, and Colloid Degeneration.

2. The Infiltrations.—These differ from the Metamorphoses inasmuch as the new material which exists in the tissues is not derived from their albuminoid constituents, but is deposited in them from the blood: there is an infiltration and deposition of a new substance. This is rarely followed by destruction of the histological elements, or by softening of the intercellular substance; hence the anatomical characters of the tissue are much less altered than in the Metamorphoses, and function is much less interfered with. The Infiltrations include Fatty, Amyloid, Calcareous, and Pigmentary Infiltration.

CHAPTER IV.

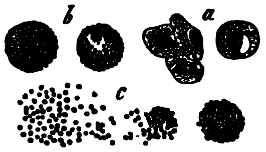
THE METAMORPHOSES.

FATTY DEGENERATION.

THIS is the most important of the degenerative processes, not only on account of its extreme frequency, but also from the deleterious effect it produces in those tissues which are affected by it.

The process consists in the transformation of the albuminoid constituents of the tissues into fat, the cells being the parts which are most frequently affected. This fat makes its appearance as minute granules and molecules within the cells, usually first in the cell-contents, and subsequently in the nucleus. The granules—which are characterized by their dark color, sharp contour, strong refractive power, and solubility in ether—gradually increase in number, until perhaps they completely fill the cell. As they increase many of them may coalesce, and so form distinct drops of fat; this, however, is not common, the fat usually remaining to the last in a granular form. As the process proceeds the cells often undergo an increase in size, and become more globular in shape, the nucleus is completely destroyed, as is also the cell-wall when this exists, and ultimately the cell is transformed into a mass of granular fat (Fig. 3).

FIG. 3.



Fatty Degeneration of Epithelium. *a.* Cells containing molecules of fat. *b.* Granular corpuscles; in one nucleus is still visible. *c.* Disintegration of corpuscles. (Rindfleisch.)

These granules of fat may remain in a coherent form for some time after the cell-wall and nucleus are destroyed; they then constitute the so-called "inflammatory" or "exudation corpuscles," or "corpuscles of Gluge," which are so common in chronic cerebral softening, and in other forms of fatty degeneration (Fig. 3 *b*). Ultimately the corpuscles break up, the albuminous matter between the granules of fat liquefies, and the fat becomes distributed in the tissue (Fig. 3 *c*).

Types of this pathological condition are furnished by many well-known physiological ones, one of the most characteristic of which is perhaps the secretion of milk. The mammary gland is a large racemose gland, consisting of innumerable groups of lobules lined with epithelial cells. The secretion of milk takes place in the following manner: The cells lining the lobules of the gland multiply abundantly, and the new cells as they are produced gradually become converted into fat; the cell breaks up, and the fatty matters in a more or less coherent form constitute the milk-corpuscles. At the commencement of the process they cohere and form colostrum-corpuscles. The milk-corpuscles thus formed are pushed forwards in the ducts of the gland by the continuous formation of new cells from below, which in their turn undergo fatty degeneration, and in this manner a continuous formation and destruction of cells takes place.

Other examples are afforded by the formation of the sebaceous matter of the skin, the cerumen of the ears, and the corpus luteum in the ovary; all of which take place in the same way by the fatty degeneration and destruction of recently-formed cells.

The immediate effect of fatty degeneration is to produce more or less softening of the affected part, the cellular elements are completely destroyed, the intercellular substance also undergoes fatty changes, and thus all trace of the original structure may be ultimately lost. This destruction of the cells is the essential feature of the change, and distinguishes it from fatty *infiltration*, in which the cells within which the fat accumulates remain intact (see "Fatty Infiltration").

If large tracts of tissue are affected, the change is readily recognizable by the naked eye, by the diminution in consistence and elasticity which are produced, and in many cases also by the opaque yellowish-white color. If, however, the change is limited to minute and isolated portions of the tissue, its existence can only be discovered with the aid of the microscope.

CASEATION.—In many cases the process of fatty degeneration is modified, and the partially degenerated tissue gradually dries up into a yellow substance of the consistence of cheese. This appears to be owing to a natural dryness of the tissue: it is most frequent in parts which contain but few vessels, or in those in which these become obliterated by some new growth. It is in growths composed of closely-crowded cells—as tubercle, epithelial accumulations within the pulmonary lobules, growths in the lymphatic glands, and in the osseous structures—that caseation is most frequently met with.

The process consists in a gradual drying up of the degenerated elements: the fluids are absorbed, the cells—which are many of them incompletely degenerated—shrivel and atrophy, the fat undergoes partial saponification, cholesterine forms, and the tissue is thus converted into a soft, yellowish-white, cheesy substance, composed of atrophied cells, fatty débris, and cholesterine crystals. This material may gradually dry up more and more, and ultimately become encapsuled by a layer of fibrous tissue, after which it remains inert in the midst of the surrounding structures.

These cheesy masses are constantly met with, especially in the lungs, and considerable confusion has arisen as to their nature and origin in this situation. This has proceeded from its having been formerly the custom to look upon all cheesy masses as essentially tubercular. Tubercle, it is true, invariably undergoes, to a greater or less extent, fatty degeneration; and it may thus, like all other structures which have undergone this process, become converted into a yellow cheesy substance; but it is by no means true that all cheesy masses are tubercular. Thus the pathological significance of these cheesy masses is much less limited than was formerly supposed; and is indeed almost coextensive with that of fatty degeneration itself. In whatever situation they are met with, they indicate merely that the histological elements have undergone this fatty metamorphosis, and under no circumstances are they in themselves evidence of any one particular form of morbid growth.

The caseous mass may subsequently become calcified, or undergo a process of softening and liquefaction.

CALCIFICATION.—This is an advanced stage of the preceding process. It most frequently occurs in those cases in which the caseous mass is completely inclosed and isolated from the external air, as when in the lymphatic glands, in bone, or when encapsuled in the lungs. The mass becomes infiltrated with calcareous particles, and is thus converted into a calcareous concretion (see "Calcareous Degeneration").

SOFTENING.—This process consists in a liquefaction of the caseous substance, which is probably owing to some chemical change in its constituents. It commonly occurs in parts which come into contact with the external air, especially in those situated in the intestine, and in the bronchial mucous membrane, and pulmonary lobules (see "Tubercle" and "Catarrhal Pneumonia"). The caseous mass liquefies, and is converted into a thin puriform fluid, containing curd-like cheesy matter, which to the naked eye looks much like pus, but under the microscope is seen to consist simply of granular débris, fat, and cholesterine crystals. This, if not discharged, may, like the caseous masses, ultimately dry up and become calcified.

CAUSES.—All those conditions which tend to interfere with nutrition, and have been already enumerated as causes of atrophy, predispose to this fatty change. It may indeed be looked upon as an advanced stage of the atrophic process.

Diminished supply of blood is a common cause of fatty degeneration. This is seen in chronic cerebral softening, which is intimately connected with the interference with the circulation, consequent upon disease of the cerebral bloodvessels. In the heart, again, this form of degeneration is not an infrequent concomitant of disease of the coronary arteries. The circulation may also be interfered with by pressure from without, as by the growth of adipose tissue between the fasciculi of muscle, in which case the muscle undergoes fatty degeneration. The degeneration of the hepatic cells in cirrhosis of the liver is due to the same cause. Inflammation, again, is a most potent cause of this degeneration. The vitality of the part is diminished by the inflammatory process, and it is ultimately destroyed by the fatty change. This, indeed, is the most common termination of inflammation, and is exemplified by its effect on nearly every organ and tissue. The vitality of a part may become exhausted by its rapid growth, and fatty degeneration thus be induced. This explains the degeneration and softening so liable to occur in all rapidly growing new formations, the softening of the central portions of cancer, sarcoma, &c., being very familiar examples. Lastly, the vitality may be impaired, as the result of old age, and fatty degeneration consequently ensue.

It will thus be seen that fatty degeneration is a common termination of many morbid processes which tend to lower the vitality of the parts affected by them; and that this impairment of vitality is the invariable antecedent of the fatty change,—the change itself merely indicating that, from some cause or other, the vitality has been diminished. The pathological significance of fatty degeneration thus becomes in many cases a question of considerable difficulty, it being often impossible, from an examination of the degenerated parts alone, to decide upon the nature of the antecedent condition. In many cases of fatty degeneration of the kidney, for example, in which the cells lining the tubuli uriniferi are destroyed, and the tubes themselves are filled with the fatty débris, it is often impossible to say, from an examination of the tubes alone, whether the fatty change is secondary or not to an inflammatory process. It is only when an investigation of the first stages of the process is practicable that the discovery of its real nature becomes possible.

With regard to the source of the fat, it is as already stated, derived directly from the tissue itself, and is not infiltrated from without. The exact nature of the change appears at present to be somewhat uncertain. It is most probable, however, that it is simply a liberation of the fat which is naturally combined with the albuminoid constituents of the tissues, and not a direct conversion of nitrogenous into fatty matter. Whether this be so or no, the formation of the fat is undoubtedly the result of impaired nutrition, and as Dr. Quain has shown, it may take place in tissues after death, from the spontaneous decomposition of their nitrogenous constituents.

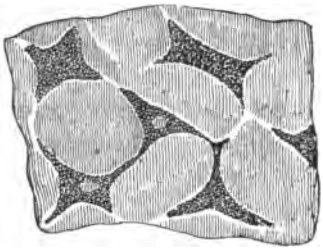
FATTY DEGENERATION OF ARTERIES.

Fatty degeneration of arteries may be a primary or secondary affection. As a secondary process it is met with in atheroma, in which the fatty change is preceded by multiplication of the cells of the sub-epithelial connective tissue (see "Atheroma").

Primary fatty degeneration is a passive process, not being preceded by any increased nutritive activity of the parts affected by it. It may affect both the internal and middle coats of the artery, but it is most common in the former situation. The usual seat of the change is the epithelial and connective tissue cells of the internal coat, small isolated groups of cells becoming affected in various parts of the vessel.

In the earlier stages of the process, when the cells are filled with fat, the condition is recognized by the existence of small irregular-shaped patches of an opaque yellowish-white color, projecting very slightly above the surface of the *intima* (Fig. 4). These may at first be mistaken for atheroma: they are in most cases, however, readily distinguishable by their superficiality, and by the facility with which they can be stripped off from the subjacent layers, which present a natural appearance. In atheroma, on the other hand, which affects the deeper structures, if the superficial layer be removed, the opacity and thickening are seen to exist beneath it. In many cases the change is limited entirely to the epithelial lining of the vessel; the more the subjacent connective tissue cells are involved, the greater is the irregularity in the shape of the patches, and the less readily can they be separated with the forceps. The opaque patches ultimately break down, the cells are destroyed, the intercellular substance softens, and the granular débris is carried away by the circulation, leaving small, irregular, superficial erosions upon the lining membrane of the vessel. These erosions are not ulcers in the true sense of that term, not being the result of an active process: they resemble the superficial erosions so common upon the mucous membrane of the stomach, as described by Dr. Wilson Fox.

Fig. 4.



Fatty Degeneration of Connective Tissue Cells in the Internal Coat of an Artery. x 300 (Rindfleisch).

Fatty degeneration may also affect the muscular fibres of the middle coat, and hence become an important element in the production of dilatation, aneurism, and rupture of the vessel.

Simple fatty degeneration may occur in any of the arteries, but it is in the smaller ones that its injurious influence is most marked. Here, by diminishing the elasticity and contractility of the vessels, it causes degenerative changes in the parts which they supply: this is exemplified by many cases of chronic cerebral softening and fatty degeneration of the heart, both of which are frequently due to this disease of the nutrient vessels. In the larger arteries, as the aorta—where it is exceedingly common—it is of less importance, the inflammatory process, atheroma, having here a far more deleterious effect.

The capillaries may also be the seat of this fatty change; the epithelial cells being destroyed in the process, and the walls so much damaged, that rupture is often the ultimate result. This is especially common in the smallest cerebral bloodvessels, where it is the most frequent cause of cerebral hemorrhage.

Passive fatty degeneration of arteries is essentially a senile change; it is an expression of that general impairment of vitality which exists in advanced life, and is usually associated with similar changes in other parts.

FATTY DEGENERATION OF THE BRAIN.

Fatty degeneration of the brain is met with in all those morbid conditions comprised under the common term of "Cerebral Softenings." Whatever impairs the vitality of the cerebral substance will tend to produce fatty degeneration, and hence softening. The portions of the brain which are the seat of this change may be merely rather softer than the surrounding healthy tissue—breaking down more readily under a stream of water which is allowed to fall upon them—or they may be completely diffluent. They are never distinctly circumscribed, but pass by insensible gradations into the neighboring tissue.

Under the microscope the change is seen to consist in a disintegration of the nerve-tissue. The medullary substance of the fibres first breaks up into large masses, and these subsequently undergo fatty metamorphosis. The tissue is thus converted into broken-down fibres, a large amount of molecular fat, and numerous large granule-corpuscles, the so-called "exudation corpuscles" or "compound inflammatory globules of Gluge." These corpuscles were formerly looked upon in all cases as the result of inflammation, hence their name; they are, however, simply conglomerations of fat-granules formed by the degeneration of the cellular elements. The cells from which they originate are, according to Virchow and Robin, the cells of the Neuroglia—the connective tissue of the brain: these cells share in the fatty change, and in doing so, appear to undergo considerable enlargement before they are destroyed, and the fatty matter breaks up. In some cases they may be seen with a cell-wall still existing, and even the nucleus is occasionally visible. They vary in size from $\frac{1}{500}$ to $\frac{1}{2000}$ inch in diameter, the average being $\frac{1}{1000}$. When the softening affects the gray matter, the nerve-cells also contain molecular fat. The small arteries and capillaries running through the softened part are many of them filled with fat-granules and granular cells: these latter probably originate in the white blood-corpuscles which have accumulated in the part and undergone fatty changes.

Molecular fat and the large granular corpuscles will also be seen adhering to the external surface of the vessels; and here care is required to distinguish these from fatty degeneration of the vessels themselves, to which the cerebral softening is so frequently due. As the process proceeds, the cerebral substance is completely destroyed, and all trace of nerve-structure is ultimately lost.

The color of the softened portion varies considerably. It may resemble that of the surrounding healthy tissue; in other cases it is altered to a yellowish or deep red tint. According to these variations in color, Cerebral Softenings have been classified into *white*, *yellow*, and *red*. The color depends in great measure upon the vascularity of the part, and on this account is important, as indicating the manner in which the softening has been brought about.

White Softening.—This is, in the great majority of cases, a chronic condition, dependent upon disease of the capillaries and small arteries which interferes with the circulation, and thus impairs the vitality of the part. There is no hyperæmia, and the color either resembles that of healthy brain-tissue, or is an opaque dirty white. White softening is sometimes acute, in which case it is due to sudden obstruction of the circulation by the impaction of an embolon, or by coagulation of the blood in one of the larger arteries.

Yellow Softening.—This is simply a variety of the former process, in which, from the fine state of division and close aggregation of the fatty particles, a dead yellowish-white color is imparted to the softened tissue. This color is probably in some cases, also partly owing to the presence of altered blood pigments, the result of some previous slight extravasation. The pigment may sometimes be seen as fine dark granules, scattered through the cells of the Neuroglia, and the nerve-cells of the gray matter, where at first sight they look like fatty particles ; they are distinguished, however, by their dark black color.

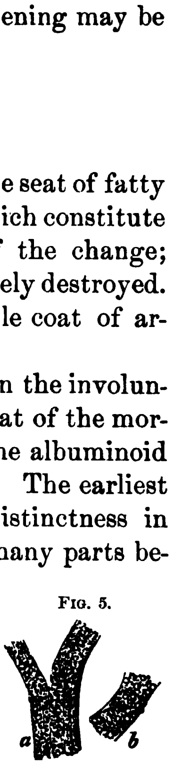
Red Softening.—This is commonly an acute affection, most frequently dependent upon vascular obstruction, either from embolism or thrombus. There is intense hyperæmia, rupture of capillaries, and extravasation of blood: the softened tissue is consequently of a deep red color: this will be described in the chapter on "Embolism." Red softening is also sometimes associated with the chronic white variety, some of the diseased vessels giving way, and thus extravasation of blood taking place into the already softened tissue. Lastly, red softening may be inflammatory (see "Inflammation of Brain").

FATTY DEGENERATION OF MUSCLE.

Both striated and non-striated muscle may be the seat of fatty degeneration. In the latter, the muscular cells which constitute the involuntary muscular fibres, are the seat of the change; they become filled with fat-granules and are ultimately destroyed. This condition is frequently met with in the middle coat of arteries which are undergoing fatty degeneration.

In striated muscle—both in the voluntary and in the involuntary of the heart—the fibres themselves are the seat of the morbid process, which consists in the conversion of the albuminoid matter of which the fibre is composed into fat. The earliest stage of the affection is characterized by an indistinctness in the transverse markings of the fibres, which in many parts become studded with minute particles of fat. (Fig. 5.) These gradually increase in number and size, and are usually distributed somewhat irregularly within the sarcolemma. In some parts single rows of granules are found running along the length of the fibre; in others, they are arranged in transverse lines corresponding with the striæ of the muscle. The fibres become extremely friable, and are readily broken up into short fragments. As the process proceeds the transverse mark-

FIG. 5.



Fatty Degeneration of Muscular Fibres of Heart. *a.* Earlier stage. *b.* More advanced. × 400.

ings entirely disappear, and nothing but molecular fat and oil-globules are seen within the sarcolemma. The sarcolemma itself may ultimately be destroyed, and nothing remain of the original fibre but the fatty débris into which its albuminoid constituents have been converted.

This is true fatty degeneration of muscle, in which the muscular elements are destroyed, and it thus differs essentially from fatty *infiltration*, in which there is simply a development of fat between the fasciculi, the fasciculi themselves not being affected (see "Fatty Infiltration").

The Heart.—It is in the heart that this condition is most frequently met with, and here it assumes a most important aspect from the deleterious influence which it exercises upon the motor power of the organ. The muscular substance may be affected throughout, or, as is more frequently the case, the degeneration may be confined to certain portions of it. It is more common in some situations than in others; the order of frequency with which it occurs in different parts is, according to Dr. Quain, firstly, the left ventricle; secondly, the right ventricle; thirdly, the right auricle; and fourthly, the left auricle.

The wider the extent of tissue that is affected, the less advanced, as a rule, is the degree of the degeneration. It is in those cases in which small tracts of tissue are involved, that the process is met with in its most advanced stage.

The consistence of the degenerated part is always diminished, and its color altered. When the change is slight, and more or less general, the muscle is somewhat softer, and more flabby than natural; it is more friable, and often breaks with a soft granular fracture. The color is uniformly rather paler, and more opaque than that of healthy cardiac tissue. Under the microscope, the muscular fibres are seen to have lost their striated appearance, and to contain minute granules of fat; there are, however, no large molecules or oil-globules, and the sarcolemma is not destroyed.

More frequent than this uniform and slight degree of degeneration affecting the whole or the greater part of the organ, is a condition in which, although the change may be more or less general, it is much more advanced in some parts than in others. In such cases the heart presents a mottled appearance; numerous opaque, pale yellowish or brownish patches are seen irregularly distributed throughout its substance. These patches vary considerably in size and form; they are met with especially in the papillary muscles, the columnæ carneæ, and in the layers of fibres immediately beneath the endocardium: they may also occur beneath the pericardium, and in the deeper portions of the organ. They correspond with the most degenerated portions of the tissue. They are soft and flabby, and have a rotten consistence, tearing readily under the finger. Under the microscope, the fibres are seen to be in the most advanced stage of fatty degeneration; their sarcolemma is filled with large molecules of fat and oil-globules, which in many parts have escaped, and lie free amongst the surrounding less degenerated tissue.

CHAPTER V.

MUCOID AND COLLOID DEGENERATION.

UNDER this head is included a class of morbid changes which are characterized by a peculiar softening of the tissues. Colloid and mucoid degeneration have frequently been described under the common term of "colloid softenings," but—although they are very closely allied and frequently associated—they appear to constitute two distinct processes; the former affecting especially the cells, the latter the intercellular substance.

MUCOID DEGENERATION.—This consists in a liquefaction of the tissues and an alteration in their chemical composition, owing to which they yield *mucin*, and become converted into a material of a soft mucilaginous jelly-like consistence. This is the condition of nearly all tissues in their immature or fœtal state: the connective tissues in the fœtus consist almost entirely of this soft mucin-yielding substance. Some tissues retain these characters after birth. The umbilical cord, and the vitreous humor of the eye, are both composed of this substance.

The change affects especially the intercellular substance, much less frequently the cellular elements. The intercellular substance of the connective tissues in their fully developed state consists of gelatin and chondrin, and the mucoid change is thus a reversion of this substance to its fœtal condition. As to the manner in which this takes place, nothing is known; it does not appear to be associated with any known general state, or with any previous change in the tissues in which it occurs. It probably depends upon some alteration in the vital performances of the cells, upon which the intercellular substance depends for its existence.

Mucin is closely allied to albumen, more so than to either gelatin or chondrin: it differs from it in not containing sulphur. Like albumen, it is only met with in alkaline fluids—being held in solution by the free alkali—from which it is precipitated by dilute acetic acid. It differs from albumen in being soluble in an excess of the acid, and also in not being precipitated by boiling, by tannin, or by bichloride of mercury: its behavior with these two reagents will also distinguish it from gelatin and chondrin, which are both precipitated by them.

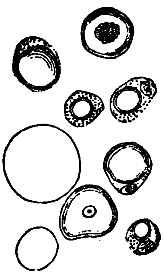
The mucoid change is by no means a common one. It is most frequently met with in cartilage, especially in the intervertebral and costal cartilages of old people; also in serous membranes, in the connective tissue of the choroid plexus and lateral ventricles, in bone, and in many of the new formations. Wherever it occurs it produces softening of the affected parts; which are transformed into a homogeneous, colorless material, of a soft mucilaginous jelly-like consistence. If the change is limited to isolated portions of the tissue, the softened parts surrounded by those which are unaltered, present the appearance of cysts. These cyst-like formations containing mucoid substance are not uncommonly met with in the costal cartilages, the choroid plexus, and in new growths.

The cells themselves appear in the majority of cases not to undergo the mucoid change; although in catarrhal conditions of mucous membranes the increased secretion of mucus is probably to be ascribed to a mucoid degeneration of the newly-formed elements (see "Inflammation of Mucous Membranes"). The cells may also be the seat of fatty degeneration.

Colloid Degeneration.—This differs from the former, inasmuch as it is the *cells* which are especially involved in the process.

The change consists in the metamorphosis of the albuminoid constituents of the cells into a substance known as *colloid* material. Colloid closely resembles mucous substance, but it differs from it chemically, in containing sulphur, and in not being precipitated by acetic acid. It is a colorless, transparent, glistening material of the consistence of jelly or half-set glue. It makes its appearance within the cells, as small lumps, which gradually increase in size, pushing the nucleus to one side, until they completely fill the cell. (Fig. 6.) The cells are thus destroyed, and converted into colloid masses. The small colloid masses subsequently coalesce, and so form larger masses of firm, transparent, jelly-like material, which are readily recognized by the naked eye.

Fig. 6.



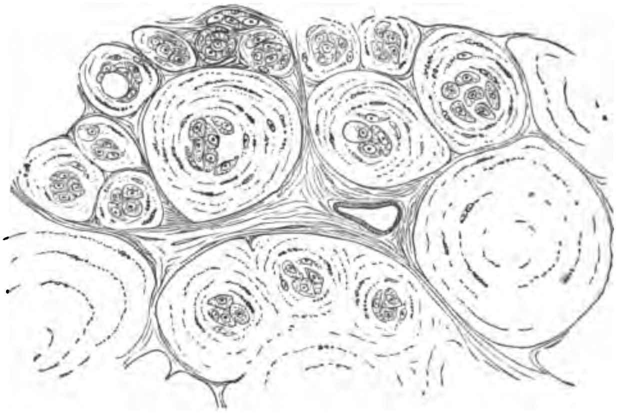
Colloid Cells, from a colloid cancer. (Rindfleisch.)

As the colloid matter increases and the cells are destroyed, the intercellular substance atrophies or softens, and in this way cyst-like cavities are formed within which is contained the gelatinous substance. Here it may subsequently undergo a process of liquefaction. (Fig. 7.)

The colloid change is most common in enlargements of the thyroid gland, in the lymphatic glands, and especially in many of the new formations (see "Colloid Cancer"). Its causes and nature are as obscure as those of the allied mucoid softening.

It is when occurring in new formations that these two forms of degeneration assume their most important aspects. Many varieties of tumors may originate as mucoid or colloid growths, or may subsequently undergo these morbid transformations. The mucous tumors (myxomas) which resemble in structure the umbilical cord, consist entirely of a gelatinous mucus-yielding substance. The sarcomas, lipomas, enchondromas, and cancers, may also become the seats of these forms of softening.

FIG. 7.



Colloid Cancer, showing cyst-like cavities filled with the colloid substance. (Rindfleisch.)

Such growths have frequently been described as gelatiniform or colloid *cancers*, this term having been applied to them without any regard to their structure or real nature. Cancers it is true may either originate as mucoid or colloid growths, or may become so after their development is completed (see "Colloid Cancer"); but it is by no means true that all tumors possessing these soft gelatiniform characters are cancers. The terms "mucoid" or "colloid" applied to a new growth, merely imply certain physical and chemical characters, and convey no information as to its real nature.

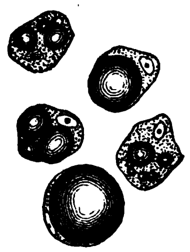
CHAPTER VI.

THE INFILTRATIONS.

FATTY INFILTRATION.

Fatty Infiltration—which is often described as "fatty degeneration"—consists in the infiltration of the tissues with fat, which is deposited in them from the blood. The fat is deposited within the cells, where it occurs as distinct drops; these may gradually accumulate and run together, displacing and obscuring the nucleus and protoplasm, until the cell is completely filled and distended with oil. (Fig. 8.) The vitality and functions of the cells are but little impaired by the accumulation, and the protoplasm—although rendered almost invisible when this is excessive—remains unaltered. This process therefore differs essentially from fatty degeneration, in which the fat is derived from a metamorphosis of the protoplasm itself, occurs in a granular form, and in which the cells themselves ultimately perish. In fatty infiltration the cells within which the fat accumulates not being destroyed, the removal of the fat is all that is necessary to restore them to their original condition.

Fig. 8.

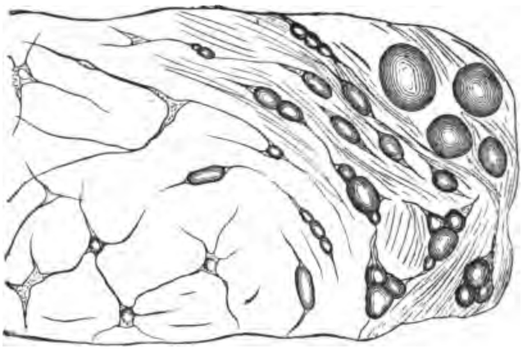


Liver Cells in various stages of Fatty Infiltration. x 300 (Rindfleisch).

The increase of fat in the blood which leads to its deposition in the tissues, is in many cases the result of excessive supply of nutriment, or to this combined with diminished waste. Such conditions obtain in people who live freely and take but little exercise. There appears to be in some a special predisposition to become fat. In other cases, the excess of fat in the blood is owing to its absorption from some particular tissue, subsequently to which it becomes deposited in another: this is seen in the fatty infiltration of the liver, which is associated with the general emaciation of pulmonary phthisis and other similar conditions. (See "Fatty Liver.") Why the deposition should take place in certain tissues, and the fat be removed from some and deposited in others, is not known.

Fatty infiltration occurs as a physiological process in the growth of adipose tissue. Adipose tissue bears a close resemblance to common connective tissue; it is connective tissue, containing numerous cells which are distended with fat. The growth of adipose tissue thus consists simply in the infiltration of more of these cells with fat. If this is excessive it constitutes obesity. (Fig. 9.) The temporary accumulation of fat in the intestinal villi and liver during the digestion of an aliment rich in fatty substances, is another example of physiological infiltration. This will be described when speaking of the "fatty liver."

FIG. 9.



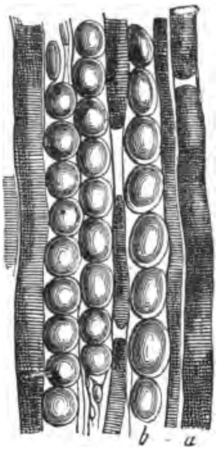
Fatty Infiltration of Connective Tissue. Showing the accumulation of fat within the cells. x 300. (Rindfleisch.)

FATTY INFILTRATION OF MUSCLE.

In muscle, fatty infiltration is frequently met with as a morbid process. The cells in the connective tissue which surrounds the fasciculi of the muscle become filled with fat; and this development of fat between the primitive muscular fasciculi has often been included under the common term of "fatty degeneration" of muscle. The process, however, differs essentially from true degeneration, in which the fat originates *within* the fasciculi and the fasciculus itself ultimately perishes, whereas in the condition now under consideration, there is a deposition of fat *between* the fasciculi, which remain—during the early stage at all events—unaffected. The interstitial fat varies in amount. In some cases single rows of fat-cells alternate with rows of muscular fasciculi, at other times the accumulation is less regular, more existing between some fibres than between others: in all cases, however, the muscular elements may be discovered lying amongst the fat. (Fig. 10.) If the latter is very considerable in amount, the muscle may appear to the naked eye to be entirely converted into fat; but the microscope will always reveal the muscular structure in which it is imbedded.

This condition is frequently met with in animals which have been fattened, the fat not only increasing in the usual situations, but also accumulating between the fasciculi of the muscles. In muscles also, which from any cause have for some time been incapacitated, this interstitial growth is extremely liable to occur; *ex. gr.* in the extensors of the wrist-joint in cases of lead-poisoning, and in long-standing paralyses from lesions in the brain or cord; also, in muscles which have been rendered useless by anchylosis of a joint. In progressive muscular atrophy, as Virchow has shown, the affected muscles exhibit this change, together with true fatty degeneration.

FIG 10.



Fatty Infiltration of Muscle. *a.* Atrophied muscular fibres. *b.* Interstitial fat-cells. x 300. (Rindfleisch.)

In the *heart*, fatty infiltration is not unfrequently met with; and here it is especially important to distinguish it from the much more common condition in which the fibres themselves are primarily affected. In health there is a varying amount of fat covering the surface of the heart, beneath the visceral layer of the pericardium, which is always most abundant in the grooves between the auricles and ventricles where it surrounds the bloodvessels. This may increase so as to completely envelop the organ, and at the same time gradually insinuate itself between the muscular fibres, so that to the naked eye all appearance of muscular structure may be lost, the walls looking like a mass of fat. In hearts less affected, striæ of fat will be seen lying amongst the muscle. The fat is always most abundant near the surface, the muscular structure becoming more evident towards the endocardium.

The immediate effect of the interstitial growth is to displace and compress the muscular fibres between which it insinuates itself, and in doing so it diminishes the contractile power of the muscle: this is especially the case when occurring in the heart. The pressure, however, which it exercises upon the fibres and the accompanying bloodvessels, ultimately causes in the former atrophic and degenerative changes. Thus the fasciculi gradually atrophy, the transverse striation becomes indistinct, and is replaced by molecular fat; in fine, true degeneration of the muscle is established. These two processes, indeed, not uncommonly go hand in hand together, the interstitial infiltration inducing the intrastitial degeneration.

FATTY INFILTRATION OF LIVER.

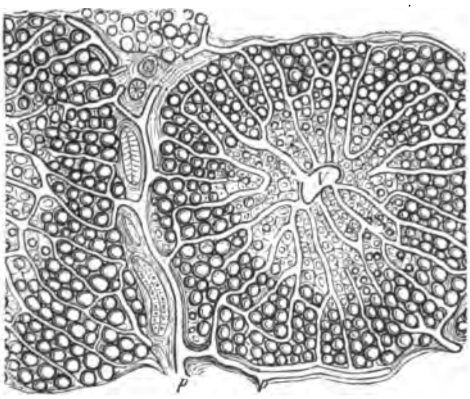
It is in the liver that fatty infiltration assumes its most important aspect, and in this organ it is exceedingly frequent, constituting what is commonly known as the "fatty liver." It must be carefully distinguished from true fatty degeneration, in which the nutrition of the hepatic cells becomes impaired, and they consequently undergo retrogressive metamorphosis. This occurs in many structural changes of the liver, as cirrhosis, amyloid degeneration, &c., and is quite distinct from fatty infiltration.

The hepatic cells always contain a small quantity of fat, which is temporarily increased after the ingestion of fatty substances. It will be well to describe this physiological infiltration before proceeding to the morbid process.

The ingestion of an aliment rich in fatty substances is followed by a temporary excess of fat in the blood, and by the deposition and temporary accumulation of a portion of this within the hepatic cells. This fat is first deposited in the cells which are in immediate contact with the capillaries of the portal vein, and thus is produced an excess of fat in the cells at the circumference of the hepatic lobules. This gradually passes from the cells at the circumference to those in the interior, whence it is ultimately conveyed into the circulation. This process goes on until the excess of fat is removed from the blood, when the hepatic cells again acquire their former character. There is thus a transitory accumulation of fat within the hepatic cells which is gradually removed, the vitality of the cells not being thereby impaired.

Precisely similar to the above is the morbid process. The morbidly fatty liver is one which contains an abnormal quantity of fat. This may result either from an excessive deposition from the blood, or from some interference with the removal of the temporary physiological accumulations. The fat being deposited from the blood in the portal capillaries, the increase is first observable in the external zone of the hepatic lobules. (Fig. 11.) It accumulates here within the cells as minute globules, which as they increase coalesce and form large drops of fat: these may ultimately completely fill and distend the cells, which at the same time become larger and more globular in shape. (See Fig. 8.) As the process proceeds, the accumulation advances from the periphery towards the centre of the lobule, until ultimately its whole mass may be involved, and the cells universally become distended with fat. The vitality of the cells is not materially impaired by the infiltration; they continue to perform their functions, as is shown by the presence of bile in the stools and in the gall-bladder.

FIG. 11.



Fatty Infiltration of Liver. (Somewhat diagrammatic.) Showing accumulation of fat in the external zone of the lobules. *V.* Hepatic vein. *p.* Interlobular portal vessels. (Rindfleisch.)

The fatty liver is somewhat increased in size, in advanced stages often considerably so. The surface is smooth, the edges are thickened and rounded, the specific gravity is diminished, although the absolute weight may be increased. If the infiltration is slight, involving merely the portal zone of the lobules, the cut surface will present a mottled appearance, the external fatty zone being of an opaque yellowish-white color, whilst the central portion remains unaltered, or is perhaps somewhat hyperæmic. The more extensive the infiltration the larger is the pale zone, and ultimately, when the whole lobule is involved, there may be left in the centre merely a reddish-brown point, which corresponds with the commencement of the hepatic vein; and in many cases even this point is lost. The organ is then of an almost uniform opaque yellowish-white color, and the boundary between the individual lobules may be completely obscured. Its consistence is much diminished, it feels doughy, and pits on pressure with the finger, and the knife used to cut it becomes coated with oil. The pressure exercised by the infiltrated fat produces considerable anæmia of the organ, but the interference with the circulation is never sufficient to cause ascites, hemorrhage, or other evidences of portal congestion.

Fatty infiltration of the liver occurs under two opposite conditions—one in which there is general obesity, and the fat accumulates in the liver in common with other parts; and another, in which there is general emaciation, and the fat is removed from the subcutaneous cellular tissue, and from the other situations in which it naturally exists, whilst it accumulates in the hepatic cells. The latter is exceedingly common in chronic pulmonary and cardiac affections, especially in pulmonary phthisis. Possibly in these cases the interference with the respiration, and the consequent incomplete oxidation of the hydrocarbons, is intimately connected with it.

CHAPTER VII.

AMYLOID DEGENERATION.

Amyloid Degeneration, which is one of the most important of the degenerative processes, consists in the infiltration of the tissues with a peculiar homogeneous translucent substance, by which their vitality becomes diminished and their functions impaired.

It was formerly known as the *lardaceous* or *waxy* change, the organs affected by it having somewhat the appearance of lard or wax. The term "*amyloid*"—which was applied to it by Virchow, from the supposed resemblance of the new material to cellulose or starch—as being that by which it is most generally known, is here adopted.

This form of degeneration is rarely a primary affection, but usually occurs in the course of some other disease. It appears to be associated with certain cachectic conditions, brought on by many exhausting diseases. It is in those diseases which are attended by profuse and long-continued suppuration, as chronic diseases of bone, empyema, chronic tubercular and other suppurative diseases of the lungs, chronic pyelitis, and syphilis, that the amyloid change most frequently occurs. In syphilis, it is those cases in which there is caries or prolonged ulceration that it is most commonly met with.

Every organ and tissue may be the seat of the change; those, however, in which it is especially prone to occur, are the liver, the kidneys, the spleen, and the lymphatic glands. It is met with less frequently in the stomach and intestines, in the suprarenal capsules, in the pharynx and œsophagus, in the bladder, prostate, and generative organs, in serous membranes, in the membranes of the brain and cord, and in muscle. It is rarely limited to one organ, but several organs are almost invariably simultaneously affected by it.

Respecting the nature of the new substance which infiltrates the tissues, the analyses of Kekulé and Schmidt show that it is a nitrogenous compound closely allied to albumen. The conclusions arrived at by these observers are, however, not satisfactory, as they were unable completely to separate the substance from the tissues in which it was deposited. More recently, Kühne has succeeded, by submitting the amyloid organs to a process of artificial digestion, in completely isolating the new material, and the results of his investigations confirm those of Kekulé and Schmidt. It appears, therefore, that the amyloid substance is an albuminoid compound closely allied to albumen, from which it is probably derived by a retrogressive metamorphosis.

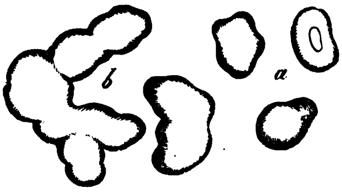
The most characteristic feature of the amyloid substance is the peculiar reaction which it gives with iodine, and with iodine and sulphuric acid. If an aqueous solution of iodine, made with the help of iodide of potassium, is applied to an amyloid organ, the affected portions change to a deep reddish-brown color; this is not permanent, but gradually passes off, and the part regains its former appearance. If the application of the iodine is followed by the cautious addition of sulphuric acid, a blackish-blue or violet tint is produced. This latter reaction, however, is not easily obtained, considerable nicety being required in the application of the reagents. The following is the method for obtaining it, recommended by Professor Virchow: A dilute aqueous solution of iodine must be allowed to soak well into the tissue, the excess must be poured off, and a single drop of concentrated sulphuric acid gradually added, when a blue or violet color will be produced, either at once or after some time. In the hands of English pathologists this latter reaction has certainly met with but little success; and if the color be obtained, it is by no means satisfactory, and more nearly resembles a black than the blue which has been described. Fortunately, however, the reaction with iodine alone is perfectly characteristic, and the attempt to obtain the blue by the subsequent addition of sulphuric acid, is therefore quite unnecessary. If the change is at all advanced, the reddish-brown color will be produced by merely pouring the aqueous solution of iodine over the cut surface of the organ; but in slighter degrees of the affection, thin sections must be made with a Valentin's knife, and well washed with water to remove the blood, before the coloration with iodine can be obtained.

Upon considering the above reactions, it will be seen that they resemble, in some respects, those exhibited by some well-known organic substances. Cellulose—the substance which forms the external membrane of vegetable cells—yields a blue when treated with iodine and sulphuric acid, and so far resembles the amyloid material; but differs in not being colored by iodine alone. The same is true of cholesterine; this also gives a reddish-brown with sulphuric acid, but is not altered by iodine. To starch, the resemblance is not so marked as to the two preceding substances: with this, iodine produces a blue color without the addition of the acid.

The amyloid substance almost invariably makes its appearance first in the small arteries and capillaries, and subsequently extends from them to the surrounding tissues. The cells of the intima and of the muscular coat are the first to become infiltrated, then the remaining structures of the artery. When the vessels have become infiltrated, the amyloid matter extends to the immediately surrounding parts; it invades both the cells and the intercellular substance, and may gradually extend until the whole organ is completely impregnated.

The changes produced in the tissues by this infiltration are very characteristic. The cells as they become filled with the new material gradually increase in size, they lose any irregularities in their contour, and become rounder and more regular in shape, their nuclei disappear, and the whole cell is converted into a structureless homogeneous body, which has a peculiar translucent glistening appearance (Fig. 12, *a*). If the cells are in close contact many of them may coalesce, and their distinctive boundaries thus become obliterated. (Fig. 12, *b*.) The intercellular substance in the same way acquires a homogeneous glistening appearance. The walls of the small arteries—in which, as already stated, the change commences—become considerably thickened, and they present the same homogeneous translucent characters. The calibre of the vessels is diminished, and the circulation through them is consequently impeded.

FIG. 12.



Liver Cells infiltrated with the Amyloid Substance. *a*. Single cells. *b*. Cells which have coalesced. x 300. (Rindfleisch.)

Organs in which this change is at all advanced, present features so characteristic that its nature can be readily recognized by the naked eye. They are usually considerably increased in size; their absolute weight is increased, and also their specific gravity; their surface is smooth, and the capsule tense and stretched; their consistence is firm and somewhat elastic. On section, they exhibit a peculiar homogeneous, glistening, translucent appearance, somewhat resembling wax or glue. Owing to the diminished calibre of their bloodvessels, and to the pressure exercised by the new material, they contain but little blood, and hence are always pale in color. The change may involve the whole organ, or it may be limited to certain portions: in the spleen, for example, it is frequently limited to the Malpighian corpuscles, and in the liver, to the cells in the immediate vicinity of the hepatic artery.

Although the above characters are often sufficiently marked, they should always be confirmed by the application of iodine to the cut surface of the organ. In slighter degrees of the affection—when the physical characters are but little altered—the application of this reagent becomes necessary in order to discover the presence of the substance. In these cases, merely pouring the solution over the organ will often fail to produce the characteristic staining; and it will be necessary to make thin sections with a Valentin's knife, and wash them thoroughly with water to remove the blood, before the reaction can be obtained.

In the earliest stages of the change the use of the microscope may be necessary for its recognition, and by this means the slightest degrees of it can be readily discovered. If thin sections of the tissue are examined, those parts which are infiltrated will present the peculiar homogeneous translucent appearance already described, the cells having lost their distinctive characters, are being united with the intercellular substance into a uniform mass. The addition of iodine will render, by the staining, any change at once apparent.

The effect of amyloid degeneration is to impair or even to completely destroy the nutrition and function of those organs which are affected by it. This is owing to two causes,—the obstruction offered to the circulation, and the direct influence of the new material upon the parenchyma of the organ. The obstruction to the circulation, which results partly from the diminution in the calibre of the small arteries, and partly from the general pressure exercised by the infiltrated substance, causes an insufficiency in the supply of arterial blood. As a consequence of this, secondary atrophic changes are induced in various parts; the cells atrophy and often undergo fatty metamorphosis, which indeed is constantly associated with the amyloid change. The infiltration of the parenchyma of the organ impairs the vitality of the cellular elements, and may ultimately completely arrest any manifestation of their functions.

As this form of degeneration is almost invariably secondary to some grave constitutional state, it can rarely be looked upon as in itself a cause of death, although it may materially hasten, and even determine the fatal termination.

Having thus described the nature of the amyloid substance, and the way in which it makes its appearance in the several tissue of an organ, it remains to consider the source from which it is derived.

The disease, as already stated, appears to be an infiltration, and to consist in the deposition of a new material from the blood in the various tissues and organs. The existence, however, of any albuminous compound in the blood which resembles amyloid in its reactions with iodine, has never been made out, even in the most marked cases of the disease. This fact must, therefore, negative the supposition that it is a simple infiltration. If derived from the blood at all, it must undergo some chemical change subsequently to its deposition in the tissues. Dr. Dickinson considers that the new material is de-alkalized fibrin, which is deposited in consequence of the loss of the alkali with which it is normally combined; this loss resulting from the profuse suppuration to which the disease is usually secondary: hence he terms it "*depurative infiltration.*"[1] Whether this be so or no, it is probable that some change in the blood is the cause of the disease, and that this latter consists in the infiltration of an albuminous or fibrinous substance which becomes consolidated in the tissues, and is not a direct metamorphosis of the tissues themselves. The way in which the several tissues of an organ are affected, the change almost invariably commencing in the small nutrient bloodvessels, and extending from them to the surrounding parts; the general character of the affection, several organs being simultaneously involved; together with the fact that it is almost invariably secondary to chronic suppurative diseases—point to some alteration in the composition of the blood as the cause of the degeneration.

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[1] "Medico-Chirurgical Transactions," vol. i.

AMYLOID DEGENERATION OF LIVER.

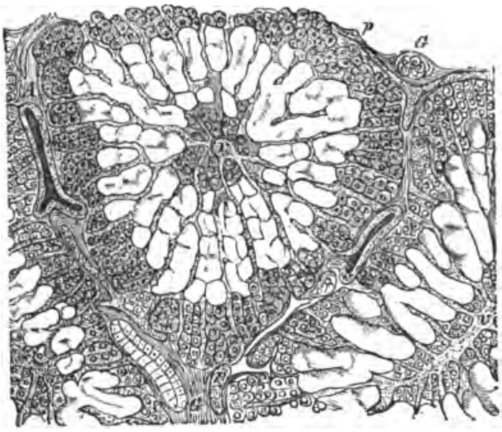
The liver is one of the most frequent seats of the amyloid change, and here, as in other parts, it commences in the small nutrient bloodvessels. The minute branches of the hepatic artery are first affected, and from these the infiltration gradually extends to the hepatic cells immediately adjacent, until ultimately the whole organ may become involved.

If a liver is examined in the early stage of the affection, and the iodine solution is applied to thin washed sections of the organ, it will be found that the characteristic staining is limited to certain portions of the lobules, viz., to those which are situated between their external and central parts. This intermediate portion corresponds with the distribution of the hepatic artery, and the hepatic cells situated between its ramifications being infiltrated with the new material before those in the external and central portions of the lobule, the brown staining is first apparent in this situation. (Fig. 13.)

The earliest seat of the amyloid change thus differs from that of the fatty. In fatty infiltration it is the most external portion of the lobule in which the fat accumulates—that which corresponds with the distribution of the portal vein (see Fig. 11). It differs also from the infiltration with pigment, which takes place in the most central portion, around the hepatic vein. Thus in each hepatic lobule three zones may be distinguished: an external one, which is the chief seat of the fatty change; a central one, which is the chief seat of the pigmentary change; and an intermediate one, which is the chief seat of the amyloid change. These three zones, indeed, may frequently be recognized by the naked eye, the pale opaque external one contrasting strongly with the intermediate one, which is translucent, and with the darker central one. In the most advanced stages of the disease, however, both the external and central portions of the lobule may become involved, and the cut surface present an almost uniformly homogeneous appearance.

The amyloid liver is increased in size, often very considerably so; it may be so large as almost completely to fill the abdominal cavity. The enlargement is uniform, and hence the natural configuration of the organ is but little altered. Its weight is increased, and also its specific gravity. Its edge is rounded, the surface is smooth, and the capsule appears tense and stretched. The consistence is firm and elastic. The cut surface is dry, bloodless, smooth, translucent, and waxy-looking, and of a pale reddish-gray or dirty yellow color. If the change is very far advanced, the tissue may be perfectly homogeneous, all distinction between the individual lobules being lost. In other cases the lobules are distinctly mapped out; they are enlarged, and the external zone may be of an opaque yellowish-white color, owing to the presence of fat. This association of the fatty and amyloid changes is exceedingly common. Amyloid infiltration does not obstruct the portal circulation, and hence does not cause ascites. It impairs the vitality of the hepatic cells, and thus interferes with the functions of the organ.

FIG. 13.



Amyloid Liver. Showing infiltration of cells in the intermediate zone of the lobule. *A.* Interlobular artery. *V.* Hepatic vein. *pp.* Portal vessels. × 300. (Rindfleisch.)

AMYLOÏD DEGENERATION OF THE KIDNEYS.

The kidneys are especially liable to be involved in the amyloid change, and are frequently the earliest seats of the morbid deposition. It may constitute in them the primary lesion, or it may occur subsequently to inflammatory conditions of the secreting and interstitial structures. As a primary change, it is one of the most important varieties of Bright's disease.

The process commences in the tufts of vessels which form the Malpighian bodies, the walls of which become thickened by the infiltration of the new material, so that the tufts are increased in size. It then proceeds to the small afferent arteries, and ultimately to the vasa efferentia, and to the arteriolæ rectæ which run through the medullary portion of the organ. After these vessels have thus become infiltrated, and before those in the pyramids are affected, the deposition extends to the intertubular tissues of the cortex, into which the amyloid substance is poured, imbedding the tubuli uriniferi in its course. In some parts it appears that the exudation takes place into the tubes themselves, as the casts which they contain are occasionally deeply stained by the iodine solution; this, however, is far from being frequently the case. In the earlier stages of the process—if the organ is not the seat of any other morbid change—the tubes and their lining epithelium present a perfectly natural appearance. Many of them contain pale hyaline casts, which also appear in the urine. These, however, are probably simply albumen and fibrin, which have transuded from the vessels; although from the reaction they occasionally exhibit, it appears that they sometimes consist of the same material as that which permeates the vessels and intertubular structures. As the change proceeds, and the new material increases in amount, the tubes become compressed, and in many places completely obstructed. If the compression is not uniform, they may dilate and form small cysts. The epithelium, which was at first normal, ultimately atrophies and undergoes fatty changes, owing to the interference with its nutrition. In some cases it appears to be the seat of a catarrhal condition, and the tubes are found blocked with epithelial products.

The first effect of this change is to obstruct the circulation in the cortex. The bloodvessels, diminished in calibre, allow little but the liquor sanguinis to pass through them, the passage of the blood-corpuscles being to a great extent prevented; hence the pallor of this portion of the organ. The arterial walls are so altered that fluids and albumen readily permeate them; and thus is produced the large quantity of urine, loaded with albumen, which characterizes the earlier stages of this affection. As the infiltration increases, and the tubes become obstructed, the urine diminishes in quantity. The excretion of urea is less interfered with than in other forms of Bright's disease, and hence symptoms due to its retention seldom occur. Tube casts are rarely numerous, they are for the most part hyaline and finely granular, though sometimes they contain fatty epithelium.

In the earlier stages of the affection, the cortex of the kidney is merely rather paler than natural, and perhaps somewhat firmer in consistence; but otherwise it presents no abnormal appearance. It is only upon the application of iodine to the cut surface, or to thin washed sections of the organ, that its diseased condition becomes evident. When this test is employed, the Malpighian bodies at once become apparent as minute red points scattered through the cortex. As the disease advances, the size of the organ increases; the enlargement, however, is confined to the cortex. The surface is smooth, and the capsule separates readily. The enlarged cortex is remarkably pale and anæmic, and has a peculiar translucent, homogeneous, wax-like appearance. Its consistence is hard and firm. A few scattered vessels may be seen on the surface, and the bases of the pyramids sometimes exhibit an increased amount of vascularity. If iodine is poured over the cut surface, the Malpighian bodies and the arteries of the cortex become mapped out almost as clearly as in an artificial injection. The enlarged Malpighian bodies may indeed often be seen as glistening points before the iodine is applied. Frequently, the homogeneous appearance of the cortex is interrupted by minute, opaque, yellowish-white lines and markings; these are produced by the fatty changes in the epithelium of the tubes, which so commonly occur in the later stages of the affection. Ultimately, the capsule may become more or less adherent, and slight irregular depressions may make their appearance on the surface of the organ: the latter are due to atrophic changes in some of the tubes. If, as is not unfrequently the case, the infiltration is associated with an increase in the intertubular connective tissue, the atrophy of the organ will be more marked. (See "Cirrhosis of Kidney.")

AMYLOID DEGENERATION OF THE SPLEEN.

Amyloid degeneration of the spleen is met with in two forms —one in which the disease is limited to the Malpighian corpuscles ("Sago Spleen"), and the other in which the pulp appears to be chiefly implicated. The former is much the more common condition. The Malpighian corpuscles are infiltrated with the amyloid substance and converted into translucent, wax-like bodies, much like boiled sago: hence its name. The process commences in the small arteries of the corpuscle, and it is only after these have become infiltrated that it extends to the corpuscles themselves. The small lymphatic cells of which the corpuscle is made up are then involved in the process, and ultimately the whole is converted into a pale, firm, translucent, glistening mass.

The sago spleen is more or less enlarged ; its weight and density are also increased. The cut surface is smooth, dry, and studded all over with small glistening sago-like bodies, varying in size from a millet to a hemp-seed, which are stained a reddish-brown color by the iodine solution. These may become so large as to occupy a large portion of the organ, although in earlier stages of the affection they are so minute that they can only be seen in thin sections of the tissue.

In the other variety of amyloid spleen, the pulpy parenchyma between the corpuscles is infiltrated with the new material. This is probably merely an advanced stage of the former condition, in which the disease extends from the corpuscles to the surrounding pulp; the whole organ being ultimately involved. Under these circumstances the organ often attains a considerable size, much larger than is met with in the sago spleen. It is remarkably hard and firm, and the capsule is tense and transparent. On section it presents a dry, homogeneous, translucent, bloodless surface, of a uniform dark reddish-brown color. Thin sections can be readily made with a knife, the organ cutting like soft wax. The corpuscles are not visible as in the former variety, being probably obscured by the surrounding pulp, which has become so densely infiltrated with the amyloid substance.

AMYLOID DEGENERATION OF LYMPHATIC GLANDS.

In the lymphatic glands the process much resembles that in the spleen. The small arteries in connection with the follicles of the gland are the earliest seats of the change; and from these it extends to the minute cells with which the follicle is filled. The follicle is thus ultimately converted into a small homogeneous mass.

The glands are enlarged, and on section the minute wax-like bodies can often be seen scattered through the cortex. The cut surface is smooth, pale, and translucent.

As these glands are largely concerned in the formation of the blood-corpuscles, their implication in the amyloid change must to a large extent aid in producing the emaciation and anæmia which characterize this affection. The same is true of the spleen, which is usually simultaneously involved.

AMYLOID DEGENERATION OF THE ALIMENTARY CANAL.

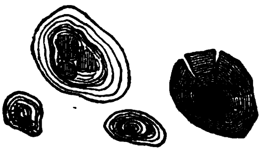
The whole of the alimentary tract may be the seat of the amyloid change, and here it assumes an important aspect from the deleterious influence which it exercises upon the absorbent and secreting processes, and from the consequent impairment of the general nutrition which results. The disease, however, in this situation is very apt to escape observation, as it produces but little alteration in the appearance of the parts. The mucous membrane may look somewhat pale, translucent, and œdematous, but otherwise to the naked eye nothing is discoverable. It is only upon the application of iodine to the washed mucous surfaces that the nature of the change becomes apparent. In the small intestine—which is perhaps the part most commonly affected—the effect of the application of iodine is very characteristic: a number of small reddish-brown points appear over the whole surface of the membrane; these correspond to the intestinal villi, the arteries and capillaries of which are infiltrated with the amyloid substance. In the stomach and œsophagus the vessels are mapped out in a similar manner by the iodine solution. The change in the intestine gives rise to serous diarrhœa, this being probably due to an increased permeability of the infiltrated walls of the vessels.

THE CORPORA AMYLACEA.

The corpora amylacea or "amyloid bodies," so frequently met with in the nervous system in the prostate, and in other parts, have usually been looked upon as more or less allied to the amyloid substance; there appears, however, with the exception of a certain similarity in their behavior with iodine and sulphuric acid, to be no connection between them.

They are round or oval bodies, formed of a succession of concentric layers, and are often changed to a deep blue color by iodine, thus bearing both in their structure and chemical properties the strongest resemblance to granules of vegetable starch. (Fig. 14.) Sometimes, however, the blue is only exhibited after the subsequent addition of sulphuric acid, and thus a resemblance is shown to the amyloid substance. They vary in size from microscopic granules to bodies which are distinctly visible to the naked eye; sometimes being as much as one or two lines in diameter. The larger ones are usually formed by the conglomeration of the smaller granules, which are often inclosed by a common envelope.

FIG. 14.



Amyloid Bodies from Prostate.

They occur especially in the nervous system of those in advanced life; the ependyma of the ventricles, the white substance of the brain, the choroid plexus, the optic nerve and retina, and the spinal cord being their favorite seats. The larger forms are met with most frequently in the prostate. The prostate of nearly every adult contains some of these bodies; and they may accumulate here to such an extent as to form large concretions. They are also occasionally met with in the lungs, and in mucous and serous membranes.

As has been said, they usually exhibit a bright blue color upon the application of iodine alone, although in some cases not until the subsequent addition of sulphuric acid. Many of them, however, are colored green, or even brown by these reagents. The green is due to their admixture with nitrogenous matters, which give a yellow color with iodine, and hence the combination yields a green. The greater the amount of nitrogenous matter the more brown does the color become.

From the laminated structure of these bodies they would appear to be formed by the gradual precipitation of some material, layer by layer, upon the surface of pre-existing particles. The nature of the material, however, appears in no way to resemble that of the substance which infiltrates the organs in amyloid degeneration. The two processes are so essentially different, both in the circumstances under which they occur, and in the characters and seat of the morbid products, that they cannot be looked upon as in any way analogous. Amyloid degeneration is a general change in which numerous organs are infiltrated with an albuminoid substance; whereas the formation of the corpora amylacea is evidently of a local nature; it is often preceded by those local atrophic changes associated with advanced life, and consists in the deposition of some starch-like material, probably liberated in the tissues themselves, upon any free body which may exist in its vicinity.

The corpora amylacea, especially those occurring in the choroid plexus and in the lateral ventricles are very liable to become calcified, and they then constitute one form of "brain sand" which is so often met with in these situations.

CHAPTER VIII.

CALCAREOUS DEGENERATION.

Calcareous Degeneration—or, as it is more commonly called, *Calcification*—consists in the infiltration of the tissues with calcareous particles. Physiologically, an infiltration of calcareous particles takes place in the formation of bone, in which lime and magnesian salts are deposited in the fibrous or cartilaginous matrix. This physiological is precisely similar to the pathological process. It is important, however, to distinguish simple calcification from ossification. In the latter, there is not only a deposition of lime salts, but an *active* change in the tissue itself—a proliferation of the cellular elements, an intimate union of the calcareous matters with the tissue, and the formation of a true osseous structure in which the calcareous particles are not visible. Calcification, on the other hand, is a purely *passive* process; there is no increased nutritive activity of the part, no multiplication of elements, no alteration of the structure, but merely an infiltration with calcareous particles.

An infiltration and deposition of calcareous substances occurs under two opposite conditions, one—in which there is an absolute increase in the amount of these constituents in the blood, and the excess becomes deposited in the tissues; the other—in which there is no such increase, but the deposition takes place owing to some alteration in the tissue itself.

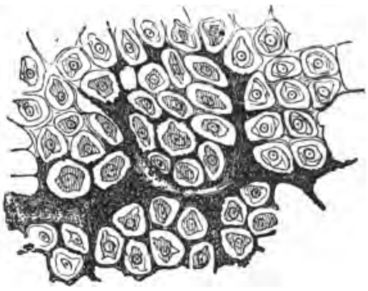
An absolute increase of the saline constituents in the blood, and the deposition of the excess in the tissues, is much the least frequent form of calcification. It occurs in some forms of softening of bone, especially in extensive caries and osteomalacia. In these diseases the lime salts are removed from the bone, returned into the blood, and deposited in other tissues. In such cases the calcification is usually more or less general—many organs being simultaneously involved. In osteomalacia, it is not uncommon to find the kidneys, the lungs, the stomach, the intestines, and even the dura mater and liver, infiltrated to a greater or less extent with lime salts. The excess of saline matters in the blood may also be due to interference with their excretion by the kidneys. In chronic structural disease of these organs, calcification may sometimes result from the consequent impairment of their excreting functions. To whichever of these causes the excess is due, the deposition will commence in the tissue immediately surrounding the bloodvessels from which the calcareous matters are derived: thus, in the lungs, the seat of the change is the interlobular tissue; in the stomach, the stroma between the glands; and in the kidney, the tubuli uriniferi and the intertubular tissue. If the process is the result of impaired excreting function of the kidneys it may be limited to them; the pelves, and the tubes of the papillæ and pyramids, being the earliest seats of the deposition. Precisely analogous to this form of calcification is the deposition of the excess of urate of soda which takes place in gout.

In the great majority of cases, however, calcification is a *local* change, depending not upon any alteration in the composition of the blood, but upon changes in the tissues themselves, owing to which some of the saline matters which are normally held in solution in the blood are deposited in them. The alteration in the tissues consists in some impairment of their nutrition, associated with a diminution in the amount of blood, and a retardation of its circulation. All those conditions which tend to produce atrophic and retrogressive changes in a part, and at the same time to interfere with the circulation in it, are liable to be followed by its calcification. Inflammation, rapidity of growth, diminished nutritive supply, and general impairment of vitality, may all of them give rise to this process. This is seen in the calcification of the vegetations in endocarditis, of the caseous masses in the lungs and lymphatic glands, of many new formations, and of the bloodvessels and cartilages in old people. In its morbid antecedents calcification thus somewhat resembles fatty degeneration, and its pathological signification is in many cases equally difficult to determine. Fatty degeneration indeed very frequently precedes the calcification, which is merely a later stage of the retrograde process.

Respecting the cause of the deposition of the calcareous substances in the atrophied structures, it is probably partly owing

—as stated by Rindfleisch—to the stagnation of the nutritive fluids in the part, owing to which the free carbonic acid which holds the salts in solution, escapes, and they are consequently precipitated; and partly to the non-assimilation of these fluids by the degenerated elements of the tissue. The calcareous particles make their appearance both within the cells and in the intercellular substance; they are much more frequent, however, in the latter situation. They are seen at first as fine molecules scattered irregularly through the intercellular substance. (Fig. 15.) They are characterized when viewed by transmitted light by their opacity, dark black color, and irregular outline, and also by their solubility in dilute mineral acids. They gradually increase in number until ultimately large tracts of tissue may be converted into an opaque calcareous mass, in which the cells are inclosed and can no longer be recognized. These larger masses have a sharp black irregular outline, and when the calcification is complete, acquire a homogeneous, glistening, semitransparent appearance. The cells themselves are much less frequently infiltrated, being usually merely inclosed and obscured by the calcified intercellular substance. Calcareous particles may, however, make their appearance in the cell-contents, and gradually increasing, convert the cell into a homogeneous calcareous body.

FIG. 15.



Calcified Cartilage. × 300. (Rindfleisch.)

The calcareous matters consist for the most part of lime and magnesian salts, especially the phosphates and carbonates. If the latter are present, the addition of a little dilute hydrochloric acid is followed by the appearance of numerous minute air-bubbles in the tissue, owing to the liberation of carbonic acid.

In those cases in which calcification is associated with retained gland secretions, the calcareous matters will consist of the specific gland salts.

A part which has become calcified undergoes no further change ; its vitality is completely destroyed, and it remains as an inert mass. In this respect calcareous differs from fatty degeneration. In the latter, subsequent changes invariably take place; the part either softening, caseating, or becoming the seat of calcification itself. It differs also in its effect upon the tissue; the structure of the affected part is not destroyed, and there is no annihilation of histological elements, such as occurs in fatty degeneration: the tissue is simply impregnated with calcareous matters, which have no other effect upon it than to render it inert; its vitality is destroyed, but its structure—in so far as the calcification is concerned—remains unaltered. If the saline matters are dissolved out with a little dilute mineral acid, the structure of the part may be again recognized, unless, indeed—as is usually the case—it has been destroyed by any antecedent change.

Calcification must thus be looked upon in many cases as a salutary lesion, the impregnation with calcareous matters preventing subsequent changes in the part. This is especially the case when it is secondary to other forms of degeneration. It is the most favorable termination of the large class of fatty changes, as is exemplified by the calcification of many inflammatory products, of tubercle, and of many other new formations. It may, on the other hand, under certain circumstances, be attended with most deleterious consequences, as is the case when it affects the arterial system.

CALCIFICATION OF ARTERIES.

Calcification of arteries may, like fatty degeneration, be a *primary* or *secondary* affection. As a secondary change it constitutes one of the terminations of the atheromatous process, and as such is constantly met with in the aorta and its branches. (See "Atheroma.")

Primary calcification is essentially a senile change, and is the result of that general impairment of vitality which exists in advanced life. It is associated with atrophy of the arterial tissues, and in some cases with more or less fatty degeneration. As it is not dependent upon a local cause, but upon general malnutrition, the change is a general one, and when occurring in one part is met with in others. It is, however, especially frequent in the superficial arteries, in the arteries of the upper and lower extremities, and in those of the brain. Its most common seat is the middle coat, where it commences in the muscular fibre cells. The calcareous particles make their appearance at first around and within the nucleus, and gradually increase until they fill the cell, which becomes converted into a small calcareous flake. The process may go on until the muscular coat is completely calcified, or it may be limited to isolated portions of the coat; giving rise to numerous calcareous plates which are irregularly distributed throughout it. From the muscular, it may extend to the external and internal coats, until ultimately the vessel becomes calcified throughout. In the majority of cases, however, the middle coat only is involved.

The vessel thus calcified, loses its elasticity and contractility; its lumen is diminished, and it is transformed into a hard, rigid, brittle tube. This condition is exceedingly common in the external iliac and in the vessels of the lower extremity, where it is a frequent cause of "senile gangrene."

CHAPTER IX.

PIGMENTARY DEGENERATION.

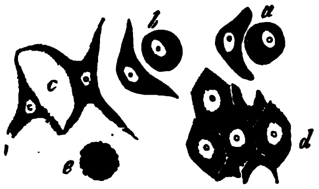
PIGMENTARY DEGENERATION, or Pigmentation, consists in an abnormal formation of pigment in the tissues. All true pigments are derived from the coloring matter of the blood. Physiologically they are for the most part eliminated by the kidneys and liver; or they may be deposited in the tissues and there remain permanent. The choroid coat of the eye and the skin of the negro are well-known examples of tissues in which there is this permanent accumulation of pigment. The cells in these situations appear to be endued with a special power to abstract the coloring matters from the blood and to store them up in their interior, where they undergo certain chemical changes and become converted into pigment.

In the pathological process also, the pigment is derived from the same source, although its presence in the tissues is rarely dependent upon any abnormal secreting powers in their cellular elements, but is the result of certain changes in the circulation or in the bloodvessels, owing to which the coloring matter of the blood escapes and infiltrates the surrounding parts. This escape of hæmoglobin may be owing to rupture of the vessels themselves, or to conditions of congestion or stasis, in which the blood-corpuscles and liquor sanguinis pass through their walls. In either case the hæmoglobin will permeate the tissues and ultimately be converted into pigment. Rupture of the vessels and the direct extravasation of blood, is, however, the most common antecedent of the pigmentary change. Soon after the extravasation has taken place, the hæmoglobin escapes from the red blood-corpuscles, either by exudation or by destruction of the corpuscle, and mixed with the liquor sanguinis, infiltrates the surrounding tissues. In other cases it escapes without any solution of continuity in the walls of the vessel. This frequently occurs in conditions of inflammatory stasis and mechanical congestion, in which serous exudation takes place into the tissues, and the hæmoglobin is liberated from the blood-corpuscles within the vessels, from which it transudes, dissolved in the liquor sanguinis. Lastly, the red corpuscles themselves may pass through the walls of the capillaries without rupture having taken place. In whichever of these ways the solution of hæmoglobin is derived, it infiltrates the tissues, staining both the cells and the intercellular substance a yellowish or brownish-red color. It is taken up, however, more readily by the cells than by the intercellular substance, or by membranous or fibrous structures; and it stains only the cell-contents, the nucleus and cell-wall remaining unaltered.

After the coloring matter has remained for some length of time in this diffuse form, it undergoes certain changes; it becomes darker and more or less granular, minute reddish-brown or black granules and crystals make their appearance both in the cells and in the intercellular substance, and these may gradually increase and form larger masses. This change in the extravasated hæmoglobin is a chemical one, and the substance into which it is converted is *hæmatoidin.* Hæmatoidin appears to be closely allied to the coloring matter of the bile, *cholepyrrhin*, which is also a derivative of hæmoglobin. It exhibits similar reactions when treated with concentrated mineral acids, displaying the same variations of green, blue, rose, and yellow colors. It is insoluble in water, alcohol, ether, and in dilute mineral acids and alkalies; it is soluble in the caustic alkalies, giving a red color. It contains more carbon than hæmoglobin; and it also contains iron.

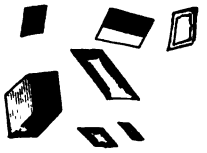
The granules of hæmatoidin vary in size from the smallest particles to masses as large as a red blood-corpuscle. The larger ones are round, or more commonly irregular in shape, and have a sharp defined border. Their color varies from yellow, red, and brown, to black. These variations appear to depend upon the age of the granules and the tissue in which they are formed; the older they are the blacker they become. The smaller granules are usually dull and opaque, the larger ones, however, often present a more or less glistening appearance. (Fig. 16.) The crystals of hæmatoidin are oblique rhombic prisms, usually of a beautiful yellowish-red or ruby-red color, sometimes approaching to brown or black. They may also occur as little plates and fine needles, but these are less common forms. (Fig. 17.) They are in most cases so small that considerable care is required to recognize their crystalline nature under the microscope, and they may easily be overlooked as merely irregular granular masses; in some cases, however, they attain a larger size. They are more or less transparent and present a shining strongly refracting surface.

FIG. 16.



Cells containing pigment. *a*, *b*, *c*, *e*. From a cancer. *d*. Epithelium from a bloodvessel. × 300. (Rindfleisch.)

FIG. 17.



Hæmatoidin crystals. (Virchow.)

Whether the hæmoglobin is converted into granular or crystalline hæmatoidin appears partly to depend upon the tissue in which it is situated, the crystals being exceedingly common in some situations, as in the brain and ovaries, whereas in others, as the lungs, only the granules are met with. Both the granules and crystals are characterized by their durability and by their great powers of resistance; when once formed they undergo no further change.

Those forms of pigment—both granular and crystalline—which are of an intensely black color, have been supposed to consist of a substance which differs in chemical composition from hæmatoidin, and which has been called *melanin*. There appears, however, to be no foundation for such a distinction; melanin is probably merely hæmatoidin which has become more or less altered by age. It is endued with greater powers of resistance, being less readily soluble in reagents than the more recently formed hæmatoidin.

Pigmentation, although one of the most common and universal forms of degeneration, is of comparatively little importance as a morbid process. The mere existence of pigment within and between the histological elements of the tissues, has in itself but little influence upon their vitality and functions. The atrophy and impairment of function which so frequently accompany it, must rather be looked upon as the result of those conditions upon which the formation of the pigment depends, than as in any way owing to the presence of the pigment itself. As evidence of other antecedent conditions, it assumes, however, a more important aspect. The pigment being derived from extravasated hæmoglobin, in whatever situations it occurs, it must be looked upon as the result of some alteration in the circulation or in the bloodvessels, owing to which the escape of the coloring matter is permitted. An exception to this exists, however, in the case of certain pigmented new formations in which the presence of the pigment appears to be partly owing to the selective power of the cells; these, like those of the choroid, separating the coloring matter from the blood. It is those growths which originate in tissues normally containing pigment, as the choroid and rete mucosum, which are most frequently melanotic.

Pigment is often the only evidence of a former extravasation. This is frequently the case in cerebral hemorrhage, where the crystals of hæmatoidin may be all that remains to indicate that rupture of the capillaries has taken place. In the ovaries, also, the slight hemorrhage which follows the escape of the ovum at each menstrual period, is marked by the formation of pigment which constitutes the "corpus luteum." In mechanical congestion and inflammation, again, the consequent pigmentation may be the principal evidence of the former existence of these conditions: this is especially seen in pigmentation of the mucous membrane of the stomach and intestines. The formation of pigment is thus, with few exceptions, the result of some antecedent change in the bloodvessels or circulation; and its presence in the tissues appears to be little more than a testimony to the existence of those processes upon which its formation depends.

False Pigmentation.—There are certain forms of discoloration of the tissues which are not due to the presence of hæmatoidin: these must be distinguished from true pigmentation. The most important of them, and that which is most closely allied to the process already described, is the staining of the tissues with the coloring matter of the bile, which is itself a derivative of hæmoglobin, and is, as before stated, very analogous to hæmatoidin. This yellow staining may affect nearly all the tissues, constituting "jaundice;" or it may occur in the liver alone, from local obstructions to the small bile-ducts, as is often seen in cirrhosis of that organ. In these cases, however, there is merely the staining of the tissues with the coloring matter of the bile, and no subsequent conversion of this into pigment.

The discoloration caused by the long-continued use of the salts of silver must also be distinguished from true pigmentation: the color here is due to the deposition of the silver in the tissues. The black color of gangrenous parts, and that sometimes produced by the effusion of large quantities of blood into the tissues, must again not be confounded with pigmentation: the discoloration in these cases is the result of the action of the sulphuretted hydrogen upon the coloring matter of the blood. The greenish-black discoloration so often seen on the surface of the liver, kidneys, and other abdominal organs after death, is in the same manner due to the intestinal gases. Lastly, the minute particles of carbon which are always met with in the lungs must be distinguished from true pigment.

PIGMENTATION OF THE LUNGS.

In no organs is pigment met with so frequently and in such large quantities as in the lungs, and here much discussion has arisen as to its nature and origin. The lungs normally contain more or less black pigment, the amount of which gradually increases with advancing age—the lungs of infants and young children being almost free from it, whereas those of adults invariably contain it in considerable quantities.

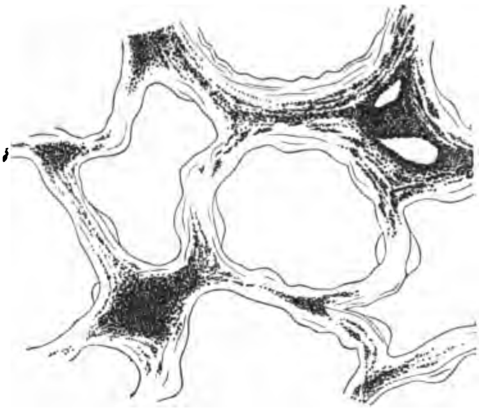
This normal pigmentation of the lungs is principally due to the presence of carbon, and not to that of true hæmatoidin-pigment. The carbon—which is derived from the incomplete combustion of wood, coal, and other substances, and is always present in varying quantities in the atmosphere—is inhaled, and the minute particles pass into the finest bronchial tubes. Having entered the bronchi, many of them penetrate the epithelial cells and mucus-corpuscles, where they may be seen as small black granules within the cells. These may readily be observed in the cells of the grayish-black sputum which is so frequently expectorated in the early morning. Much of the carbon thus inhaled is eliminated by expectoration; many of the particles, however, pass into the air-vesicles, and here their removal by this means being less readily effected, they penetrate the pulmonary substance, and make their way into the interlobular and peribronchial tissue. It is in these situations that most of the pulmonary pigment is found, and there it may be seen either within the connective tissue-cells, or lying free amongst the fibres.

As to the means by which the particles of carbon penetrate the walls of the air-vesicles and minute bronchi, nothing is certainly known. They may possibly be conveyed by amœboid cells, or by the extra-vascular nutrient fluids;—be this as it may, there can be no doubt that the penetration takes place, this having been conclusively demonstrated by physiological experiment. When once the carbon has made its way into the interlobular tissue, some of it is taken up by the lymphatics and is deposited in the bronchial lymphatic glands, where the black particles are also visible.

Closely allied to this physiological pigmentation of the lung from the inhalation of carbon, are those morbid conditions which result from the inhalation of particles of coal, stone, iron, and other substances,—of which the lungs of miners, stone-masons, and grinders afford frequent examples. Here also minute particles enter the bronchi, penetrate the pulmonary substance, and are deposited principally in the interlobular tissue. In the case of miners—in which this is most common—the particles of coal enter the lungs in such large quantities as to give to them a uniform dark black color, and like the carbon normally inhaled they are most abundant in the interstitial tissue. (Fig. 18.) In stone-masons, grinders, &c., the lungs also become deeply pigmented, although to a less extent than those of miners.

The black color of the lungs in these cases, however, is not entirely due to the presence of the inhaled substances, but partly to that of true hæmatoidin-pigment. The inhalation of the irritating particles sets up inflammatory changes in the bronchi and pulmonary tissue, causing chronic bronchitis, chronic lobular pneumonia, and a large increase in the fibrous tissue of the lungs, which thus ultimately become consolidated, excavated, tough and fibrous ("Colliers" and "Knife-grinders' Phthisis," &c.) Owing to these structural changes there is a considerable escape of coloring matter, either from rupture of the capillaries or transudation of serum, and hence a large formation of true pigment; and to this true pigment much of the dark color of these lungs must undoubtedly be ascribed. The lungs of stone-masons and grinders are like those of miners deeply pigmented, although to a less degree, but the black color in the former cases cannot be entirely accounted for on the supposition that it is due to the presence of inhaled particles.

FIG. 18.



Collier's lung, showing particles of carbon in interstitial tissue. × 300. (Rindfleisch.)

Pigmentation of the lungs from the presence of hæmatoidin occurs as the result of many other morbid conditions, many diseases of these organs being attended by the formation of pigment. In chronic phthisis, pigmentation occurs, partly as the result of the inflammatory process, and partly from the obstruction of the vessels caused by the new growth: lines of pigment are constantly seen surrounding the nodules of consolidation. In mechanical congestion also—such as frequently results from insufficiency and stenosis of the mitral orifice—the same abnormal pigmentation is seen. In these latter cases the lungs often become extremely pigmented and present a uniform brownish-red color mottled with black. The pigment is situated not only in the interlobular tissue—which is usually increased in amount—but also in the epithelium of the air-vesicles and bronchi. This condition constitutes the "*brown induration*" of Virchow and Laennec.

In acute croupous pneumonia the blood which is extravasated into the interlobular tissue and air-vesicles, and which in the early stages gives to the expectoration a rusty or prune-juice color, subsequently becomes converted into pigment, and the sputum becomes of a grayish-black; the pigment granules being visible in the newly-formed epithelial cells and mucus-corpuscles.

Pigment in the lung usually occurs as black irregular granules; it is rarely met with in a crystalline form. In all cases in which it is found in any quantity in the lung, it is also found in the bronchial glands. It is taken up by the lymphatics and, like the inhaled carbon, it becomes arrested in its passage through these glands, where it remains permanently.

CHAPTER X.

NUTRITION INCREASED.

THE morbid changes thus far described, have been attended either by *arrest* or by *impairment* of nutrition;—those remain to be considered in which the nutritive activity is *increased.* They include Hypertrophy and the New Formations.

HYPERTROPHY.

Hypertrophy is an increase in the amount of a tissue, resulting from an increase in its functional activity. The increased functional activity, and the consequent hypertrophy, are in most cases *conservative* in their nature, being induced by some necessity for an increased manifestation of the functions of the tissue; in muscle, for example, in order to overcome some obstruction; in a secreting organ, to secrete more fluid.

It is in muscular tissue that hypertrophy is most frequently met with. Examples of it are furnished, by the hypertrophy of the muscles of the calf in ballet dancers, of the left ventricle of the heart in aortic and mitral regurgitation; also by the hypertrophy of a hollow viscus, from obstruction to the exit of its contents—as of the heart from obstruction at the valvular orifices, or in the course of the circulation, of the muscular coat of the stomach in stricture of the pylorus, of the intestine above a permanent stricture, and of the bladder in stricture of the urethra. The kidney also may become hypertrophied, owing to the loss or incapacity of its fellow. In bone, hypertrophy of the fibula has been observed in cases of disease, or of ununited fracture of the tibia. In all these cases the necessity for increased activity leads to increased development.

The term "hypertrophy" is frequently applied to those enlargements of organs which result from long-continued irritation, or from other unknown causes; as to enlargements of the lymphatic glands, of the tonsils, the thymus, and of the prostate gland. Such growths, however, are some of them inflammatory in their nature, others come more properly within the category of "tumors." It is better, in order to avoid confusion, to limit the use of the term "hypertrophy" to those conservative growths which result from increased functional activity of the tissue.

The increase in the amount of the tissue may be owing simply to an increase in the *size* of the elements of which it is composed: it is then termed *simple* hypertrophy. The process, however, is usually more complex, the increased nutritive activity of the elements which leads to an increase in their *size*, leads also to an increase in their *number*, and to the formation of a new tissue, which is similar to that from which it originated: this is termed *numerical* hypertrophy, or *hyperplasia.* The two forms of hypertrophy are thus comparable with the two forms of atrophy: in simple hypertrophy as in simple atrophy, there is merely an alteration in the *size;* in numerical—an alteration in the *number* of the elements.

CHAPTER XI.

THE NEW FORMATIONS.

INCREASED nutritive activity of a tissue—as has been seen in the preceding chapter—leads not only to the enlargement of its component elements, but also to the production of new ones. It is this production of *new elements*, and the various structures to which they give rise, that constitutes the New Formations.

The New Formations comprise both *inflammatory* and *non-inflammatory* growths. Of these, the latter—which include the various kinds of tumors, together with some growths to which the term " tumor " is not strictly applicable—will be considered in the present part of this work, whereas the inflammatory growths will be treated of in the chapters on " Inflammation."

Tumors,—which constitute the greater portion of the non-inflammatory formations—are growths which, having attained a certain size, either remain permanent, or, more frequently, *tend continuously to increase.* In their development and growth they are characterized by their *independence* of the rest of the body; they increase in size by virtue of their own inherent activity, which differs from, and is independent of, that of the surrounding tissues.

There are, however, some non-inflammatory growths which cannot be regarded as " tumors " in the ordinary acceptation of that term. Amongst these are the *numerical hypertrophies*, described in the preceding chapter, and many of the *general hyperplasias* of the fibrous tissue of organs, as some forms of cirrhosis. The characteristics of the tubercular and syphilitic growths again, differ in some respects from what have been above described as those of tumors.

The inflammatory new formations differ from the non-inflammatory in being caused by some *irritation*, upon the removal of which the process of growth usually ceases. They are characterized by their *instability;* they either rapidly undergo retrogressive changes, or at the most, form a tissue which in its structure and life is similar to that from which they originated. Their tendency is gradually to approximate to a healthy condition, and, not like that of the tumors, continuously to increase and deviate from the normal type.

Whatever be the nature of the new formation, it is always the direct product of the elements of a pre-existing tissue, these elements being in some cases migrated white blood-corpuscles. In order, therefore, to understand the pathology of the new formations, it is necessary to be intimately acquainted with the histology and mode of development of the normal tissues.

ETIOLOGY OF THE NEW FORMATIONS.[1]—As the new formations are the result of the increased nutritive activity of the elements from which they originate, it will be readily understood that their causes must for the most part be obscure, and that in many cases all that can be said is, that the new growth is the result of the *spontaneous* activity of the elements from which it springs. Sometimes, however, the causes are either wholly or partially ascertainable. They may be divided into *constitutional*, *predisposing*, and *direct exciting*, causes.

Constitutional Predisposing Causes.—That many growths owe their origin to some constitutional taint, has long been a universally accepted pathological doctrine. The constitutional cause has frequently been regarded as a general one, consisting either in some alteration in the constitution of the blood, or in some abnormal condition of the physiological processes throughout the entire organism. It was formerly supposed that many new formations were the result of an exudation from the bloodvessels, and that the elements of the growth were produced spontaneously in the exuded structureless blastema. Such growths were looked upon as the local expression of a vitiated constitution of the blood—a *dyscrasia.* Although this hypothesis is now universally abandoned, and all new formations are known to originate from pre-existing cellular elements, an alteration in the constitution of the blood, or in the performance of the physiological processes throughout the body, is still regarded by many as playing an important part in their causation.

It is the malignant new formations which are thus supposed to owe their origin to the existence of a *general* constitutional taint. These growths are said to be of a *constitutional* origin, in contradistinction to the non-malignant growths, which are looked upon as purely *local*. This hypothesis is principally based upon the clinical characteristics of the malignant growths, their tendency to recur after removal, their multiplicity, and the difficulty or impossibility of completely eradicating them.

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[1] This, together with the remainder of the present chapter, applies only to the *non-inflammatory* growths.

It is said that the development of malignant growths is frequently preceded by an unhealthy state of the constitution—a cachexia. In the majority of cases of cancer, however, there is no evidence of any such cachexia preceding the local growth. The individual is usually in good health at the time of the occurrence of the primary tumor. The general impairment of nutrition and emaciation, which constitute the cachexia, are *secondary* to the local growths, and are in direct proportion to their extent and situation; the more extended the local lesions, the greater the amount of discharge, the more the lymphatics and the digestive organs are involved, the more marked is the attendant cachexia.

The multiplicity of malignant growths is also adduced as an argument in favor of the existence of a general constitutional taint. The fact that malignant growths are frequently multiple; constitutes in itself no ground for such a conclusion. The multiplicity is for the most part a *secondary* phenomenon, the secondary tumors resulting from infection by the primary one. Simple *primary* multiplicity is not so characteristic of malignant, as of many other tumors—the lipomata, fibromata, sebaceous tumors, and warts, are all more often *primarily* multiple than cancer. Multiplicity in many cases is evidence rather of a *local* than of a general taint. In scrofula, for example, the lymphatic glands generally are prone to become the seats of new growths, and tumors are often multiple in bone without occurring in other tissues. In such cases there would appear to be a *local* rather than a *general* cause.

The recurrence of the malignant growth after removal, at the seat of the operation, may again be owing to the removal having been incomplete, some of the proliferating matrix or of the growth itself having been left behind. Those growths which are essentially malignant, possess the property of infecting the adjacent tissues. This infecting power extends in many cases for some distance beyond the confines of the growth, and as the physical characters of the infected tissues differ in no way from those of the healthy, there are no means of determining how far wide of the tumor the incision must be carried in order to include the whole of the infected structures. A tendency to local recurrence is a property possessed by many growths, and it can be explained on local grounds without the necessity of admitting the existence of a general taint.

Lastly, the fact that malignant growths are usually followed by the development of similar growths in the lymphatic glands and in internal organs, may in most cases be more readily explained—as will be seen when speaking of "malignancy"—by regarding these as the result of infection by the primary tumor, than by ascribing them to the existence of a common constitutional cause.

The reasons already adduced in a preceding chapter (see "Introduction") for considering all changes in the constitution of the blood as secondary to local causes, would appear of themselves to be sufficient to render untenable the hypothesis of a *primary* blood dyscrasia. Any abnormal condition of the blood which may be associated with the development of malignant tumors must probably be regarded as resulting either from the absorption of deleterious substances, from the entrance into it of the elements of the growth, from the drain of an attendant discharge, or from interference with the processes of digestion, assimilation or secretion, or with the formation of the blood itself.

Although there would thus appear to be no necessity to admit the existence of a general constitutional cause in order to explain the clinical characters which constitute malignancy, there can be no doubt that many growths, both innocent and malignant, have a constitutional origin. The possibility of the existence of such a constitutional cause, must therefore be borne in mind in accounting for the development of secondary malignant growths, as although these may result from infection, they may also owe their origin to the same causes as those which induced the primary one. Whether the constitutional cause is a *general* one—consisting in some specific peculiarity of the entire organism, or whether it is *local* in its nature—and consists in some constitutional peculiarity of the tissues from which the new growths originate, some peculiarity which renders them more prone than other tissues to undergo abnormal development—is unknown; although the latter supposition seems to be the more probable one.

The influence of hereditary predisposition upon the occurrence of many growths, appears to point to the existence of a *local*, rather than to that of a *general* constitutional taint. In scrofula, for example, which is a markedly hereditary disease, the tendency of the lymphatic glands to undergo excessive development from very slight degrees of irritation is probably to be regarded as owing to a predisposition of the glands themselves, and not to any *general* constitutional state. The same is probably true of many other constitutional tendencies. Nævi, tumors of the skin, uterus, mammæ, stomach, and of other parts, are again all unquestionably sometimes hereditary; and here also the tendency would appear to consist in a predisposition of the tissues themselves to become the seats of new formations.

The tendency sometimes observed in *particular* tissues to generate new formations, points again to a local cause. The osseous system, for example, may be the seat of new growths—tumors occurring in nearly all the bones, and not being met with in other parts. Tumors may, in the same way, be multiple in other tissues.

Direct Exciting Causes.—The existence of a direct exciting cause is more capable of demonstration than a constitutional one. It consists either in some direct irritation of a tissue, which is by this means stimulated to increased development, or in the migration or transmission of elements from some primary growth, which, by proliferating in the tissues in which they lodge, constitute the centres of secondary formations.

The direct irritation of a tissue may be owing to simple *mechanical* or *chemical*, or to *specific* irritants. Simple mechanical or chemical irritation can, however, under no circumstances be the *only* cause of the development of the growth. The effect of such irritations alone, is to cause an inflammatory formation; in order for them to produce a non-inflammatory one, a tumor, there must be some special predisposition of the tissue itself; the irritation can merely *determine* its development. The influence of simple irritation in the production of new formations is exemplified by the frequent occurrence of epithelioma on the lips of smokers from the irritation of the pipe; and on the penis and scrotum of chimney-sweepers from the irritation of the soot ("chimney-sweep's cancer"). The numerous recorded instances of the development of a tumor following some external violence or injury, leaves little doubt that these also sometimes stand to one another in the relation of cause and effect. Lastly, it is those organs which are most exposed to irritation from external causes, as the stomach, the uterus, the mammary gland, and the rectum, which are especially liable to become the seats of new growths.

In other cases the irritant is *specific*, and induces specific changes in the tissues. It may either be introduced from without, or originate from some morbid product within the body. As to its nature, it is probably in most cases matter in a state of extremely fine division, and not in a state of solution. Such specific irritants can only act upon the tissues through the medium of the blood, lymph, or nutritive fluids. The dissemination of cancer, and of other malignant growths, is probably thus partly owing to the transmission of particles of the growth, which cause the tissues with which they come in contact to generate similar formations. Here also, however, there may exist some special predisposition of the tissues themselves.

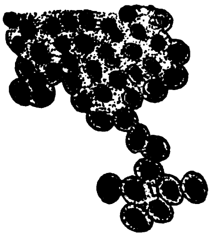
The development of new formations from cellular elements which have either emigrated, or been conveyed from their original habitat, constitutes the most frequent and important mode by which secondary malignant growths originate. The elements which may either migrate spontaneously from the primary growth, or be carried by the blood or lymph streams, proliferate in the tissue in which they lodge, and so develop into secondary formations. It is possible also that by their mere *contact* with the adjacent elements, they may induce in these similar development. This mode of origin of new formations is exemplified by the history of many malignant growths, and by this means these are most frequently disseminated. It will be more fully described when speaking of "Malignancy."

DEVELOPMENT.—The tissues from which new formations most frequently originate are the connective tissues, including under this head lymphatic (adenoid) tissue. It is especially the lower members of this class—common connective and lymphatic tissue, which are prone to generate new growths; the higher members

—cartilage and bone, are much less so. Next in order of frequency must be placed the epithelial tissues; and lastly, the higher tissues—muscles, bloodvessels, and nerves, from which the development of new growths is comparatively rare. It is probable also that many of the elements from which new growths originate—especially those of the connective tissue class—are migrated white blood-corpuscles. The part, however, which such migration plays in the development of the new formations is at present unknown.

The first stage in the process of development consists in an increase of the protoplasm of the cells, and the division of their nuclei. This is followed by multiplication of the cells, either by simple division, gemmation, or endogenous growth; and in this way a number of new cells are produced (see "Genesis of Cells"). In the case of common connective tissue—in which, as already stated, these processes most frequently occur—if the development is rapid, the early stage of cell-proliferation is precisely similar to that which takes place in the formation of an ordinary granulation. In place of the connective tissue cells innumerable small round cells are produced, from $\frac{1}{1800}$ to $\frac{1}{2500}$ of an inch in diameter, usually inclosing a large round ill-defined nucleus, which is often only visible after the addition of water or of acetic acid. These cells possess no limiting membrane, but consist of little masses of protoplasm which are almost in close contact with one another. They are distinguishable from the cells of a granulation, and from many of those met with in the rapidly growing connective tissue of the embryo. (Fig. 19.) It is from these cells, which are usually known as *indifferent* cells, that the new growth is produced. It is probable that many of these so-called "indifferent cells," do not originate from the cells of the connective tissue, but are migrated white blood-corpuscles. How far such is the case is, as already stated, not known.

FIG. 19.



Cells from a Granulation. (Rindfleisch.)

There is thus in the first place, a conversion of the connective tissue into a granulation or embryonic tissue, and subsequently of the embryonic tissue into that of which the new growth is composed. It is often impossible to determine in this early stage of the growth what it will ultimately become, whether a fibroma, a sarcoma, or an enchondroma, &c. In those growths which originate from the higher connective tissues, and from epithelium, the first stage is in the same way one of cell-proliferation, and here also, if the process is rapid, the young tissue may only subsequently assume the characters which are peculiar to the permanent growth.

The second stage of the process consists in the development of these young cells into the tissue of the permanent growth. They may form a tissue precisely similar to that from which they originated; *e. g.*, when originating from connective tissue, they may produce connective tissue; this is seen in the formation of a fibrous tumor: or when originating from epithelial tissue, they may produce epithelium, as in the formation of a glandular tumor. In other cases they produce a tissue which differs more or less from the parent tissue, as when from connective tissue springs a cartilaginous tumor, or from epithelial tissue a cancer. In all cases, however, the new growth resembles in its structure and mode of development some physiological tissue, either in its embryonic or fully developed state, so that every pathological growth has its physiological prototype. What determines the ultimate development of the young cells, why they produce such various forms of growths, is as far from our knowledge as what determines the ultimate destination of the cells in the embryo.

The whole of the primary cells may form the same kind of tissue, in which case the growth will possess the same characters throughout; or it may be complex, some cells forming one kind of tissue and some another. A combination of two or more kinds of structure may thus be met with in the same tumor—as a combination of sarcoma and lipoma, of enchondroma and myxoma, and so on.

According to the similarity or difference which subsists between the new growth and the tissue from which it grows, new formations are divisible into two classes—*homologous* and *heterologous.* When the growth resembles in its structure and development the tissue from which it originates, it is said to be *homologous*, when it differs it is said to be *heterologous.* A cartilaginous tumor, for example, growing from cartilage, is homologous, but growing from any other tissue, as from the parotid gland, it is heterologous. The same variety of tumor may thus be in one case homologous, in another heterologous. A purely homologous growth is therefore simply a *hyperplasia*, an excessive growth of a tissue in some particular part; any deviation from the type of the parent tissue constitutes heterology.

Heterology, however, is not limited to the production of a tissue which is dissimilar from that from which it originates; a growth is also said to be heterologous when it differs from the tissue in which it is *situated*, and this may occur without its being the direct product of the latter. It is heterology in this sense, that is so characteristic of the epithelial cancers. Those which originate from epithelium become heterologous, owing to the growth and extension of the epithelium beyond its normal limits (see "Epithelioma"). The same form of heterology obtains in the case of growths originating from elements which have migrated or been carried from their original habitat, and have developed into a tissue which differs from that in which they are situated.

Heterology as a rule is an evidence of malignancy. All heterologous growths, however, are not malignant, slight deviations from the normal type being no evidence of malignancy. A purely homologous growth is never malignant. A growth primarily homologous may subsequently become heterologous; this is seen in the case of a long-standing innocent tumor suddenly exhibiting malignant characters. A knowledge of the homology or heterology of a growth, is therefore an important element in the determination of its innocent or malignant nature.

RELATION OF THE GROWTH TO THE SURROUNDING TISSUES.—The relation of the tumor to the surrounding structures will depend upon its mode of growth. Growth may take place simply by the continuous proliferation of the cells of which the tumor is composed, in which case it will merely displace the surrounding parts. Having attained a certain degree of development, a fibrous capsule is often formed around it, by which it becomes completely isolated. The lipomata, fibromata, and enchondromata are usually thus encapsuled. In other cases, growth takes place also at the circumference, by the continuous proliferation of the parent tissue. There is then no line of demarcation between the tumor and the matrix from which it grows, so that although to the naked eye it may appear separate, the microscope will discover in the adjacent matrix elements of the new growth.

This is very common in the sarcomas, and in other tumors of the connective-tissue class, and is a common cause of recurrence after removal. Lastly, the tumor may increase not only by the proliferation of its matrix, but also by invading other structures;—this occurs in those growths which are essentially malignant (see "Malignancy").

RETROGRESSIVE CHANGES.—The development of the growth being complete, it becomes sooner or later the seat of retrogressive changes. The time at which these commence, varies;—as a rule the permanence and durability of a tumor bear an inverse relation to the rapidity of its growth, and to the inferiority of its organization. The more rapid the growth, and the more lowly organized the tissue formed, the less its durability and the sooner do retrogressive changes occur. The cancers and sarcomas, for example, which develop rapidly, and consist for the most part of cells, quickly degenerate; their elements are unstable and soon perish. Osseous tumors, on the other hand, which develop more slowly, and consist of a more highly organized tissue, have a much greater stability, and are but little liable to retrogressive metamorphosis.

The retrogressive changes are similar to those met with in the physiological tissues. Impairment of vitality is followed by fatty degeneration and its various terminations,—softening, caseation, and calcification. Pigmentary, colloid, and mucoid degeneration may also occur. New formations may also become the seats of an inflammatory process.

MALIGNANCY.—This consists essentially in the property possessed by many growths of infecting adjacent or distant tissues in such a way as to cause in them the production of formations *similar* to the original growth. Some growths possess this property in a higher degree than others, so that there are different degrees of malignancy. It is important not to confound the terms "malignancy" and "cancerous." "*Malignancy*" is a purely clinical term, and though in a high degree the property of the cancers, is by no means confined to them; the sarcomas, for example, are in many cases equally, or even more malignant. The term "*cancer*," on the other hand, is used to imply a definite structure, and as such is applied to a certain class of new formations.

A growth may infect other tissues in the three following ways:

1. *By its Direct Influence upon the Adjacent Structures.*—This takes place either by the nutrient fluids of the growth infiltrating the surrounding tissues and causing them to undergo similar development; or—which is probably much more frequent—by the migration of elements from the growth and their proliferation in the adjacent structures. This has already been described when speaking of the causes of the new formations. In many cases the infecting substances do not act upon the tissues immediately adjoining the malignant growth, but on those at a distance, causing a series of secondary growths in the neighborhood of the primary one. Such must be owing to the migration of cells, and not to the infiltration of juices.

2. *Through the Medium of the Lymph.*—In this case it is the lymphatic glands which are first affected. Infecting materials—consisting either of cellular elements, solid particles, or fluids, are taken up from the malignant growth by the lymphatics, and being arrested in the nearest glands cause in them similar development. When these have themselves developed into secondary growths, they in their turn constitute new centres of infection, and may thus infect more distant glands, or the immediately adjacent tissues.

3. *Through the Medium of the Blood.*—This is usually the terminal process in the history of malignant growths. The blood, like the lymph, becomes contaminated by the primary tumor, from the entrance into it either of solid matters in a state of fine division, or of cellular elements. Sometimes the walls of the vessels are destroyed by the growth ulcerating through into their interior, portions of it may then be carried off and disseminated by the circulation.

The secondary growths occur as a rule in those organs through which the blood from the primary one first passes—that is, in those organs which present the first set of capillaries either for the arrest or absorption of the infecting materials. In malignant disease of those organs, for example, which return their blood through the portal vein, as the stomach and mesenteric glands, it is the liver in which the secondary growths usually first occur, and when this has become involved, it may constitute a secondary centre of infection, and in the same way cause tertiary growths in the lungs. Although this sequence is the rule, there are numerous exceptions. In many cases the organs which are nearest in the course of the circulation to the primary growth escape, whilst those more distant become affected. This may be owing to one organ being more predisposed to the influence of the infecting materials than another; or to the capillaries of the proximal organ allowing infecting particles to pass through them, whereas those of the more distant one are small enough to arrest them. Lastly, it must be borne in mind, that the secondary growths may be entirely independent of the primary one, their origin being due to the same cause.

The evidences of malignancy in a growth thus consist—in its invasion of the surrounding structures, the implication of the neighboring lymphatics, and the occurrence of similar growths in internal organs. As a general rule it may be stated that the more juice a growth contains, and the richer it is in bloodvessels and lymphatics, the more quickly will it infect the lymphatic glands, and internal organs; on the other hand, the poorer it is in bloodvessels and lymphatics, the more are its infecting properties confined to the neighboring tissues.

The determination of the innocent or malignant nature of any growth will principally depend upon its microscopical characters, as by these alone can be certainly ascertained to which class the growth belongs. Many varieties of new formations are invariably malignant, as the cancers and sarcomas; hence any growth which from its minute structure must be included under these heads, must be regarded as being of a malignant nature. The probable degree of the malignancy of any particular tumor must be determined from its situation and mode of growth, from its succulence and vascularity, and from the clinical peculiarities of the individual case.

Classification.—New formations may be classified upon an anatomical, a physiological, or a clinical basis. Although a physiological and clinical classification are much to be desired, in the present state of our knowledge they must be very incomplete. A classification of the new formations according to their histological characters is consequently here adopted. Such a classification may be most advantageously made in accordance with the classification of the physiological tissues:

CLASSIFICATION OF THE NEW FORMATIONS.

Type of the Connective Tissues.

Type of fully developed connective tissue, ...	Fibroma.	
" embryonic connective tissue, ...	Sarcoma.	
" granulation tissue, . . .	Gumma.	
" mucous tissue, ...	Myxoma.	
" adipose tissue, ...	Lipoma.	
" cartilage, ...	Enchondroma.	
" bone, ...	Osteoma.	
" lymphatic (adenoid) tissue,	The Lymphomata,	Simple Lymphoma. Leukæmia. Tubercle.

Type of the Epithelial Tissues.

- Papilloma.
- Adenoma.
- The Carcinomata,
  - Scirrhus.
  - Encephaloid.
  - Epithelioma.
  - Colloid.

Type of the Higher Tissues.

Type of muscle, ...	Myoma.
" nerve, ...	Neuroma.
" bloodvessels, ...	Angioma.

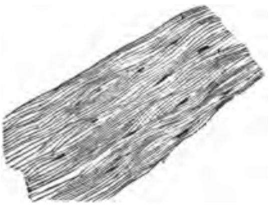
CHAPTER XII.

THE FIBROMATA.

THE Fibromata, fibrous, or connective-tissue tumors, are tumors consisting of fully developed connective tissue.

STRUCTURE.—In structure the fibromata present the same variations as those met with in connective tissue. Some of them are composed of firm, dense, fibrous tissue, such as constitutes tendons; others are laxer and less fibrous in consistence, more resembling the connective tissue of the cutis. The fibres (Fig. 20), which constitute the chief part of the growth, may be closely interlaced without any definite arrangement, or they may be grouped in bundles of various sizes: they are frequently arranged concentrically around the bloodvessels. The cells, or as they are usually called, the connective-tissue corpuscles, are very few in number, and usually only become visible after the addition of acetic acid. They are minute, spindle-shaped, fusiform, or stellate bodies, the latter having processes of varying length, which communicate with similar processes from neighboring cells. They contain in some cases an oval nucleus. The size and number of these cells varies with the rapidity of growth—the slower the growth the more fibrous the tissue, and the smaller and less numerous are the cells

FIG. 20.



Fibrous Tumor. x 200.

The fibromata usually contain but very few bloodvessels. In some cases, however, these are more numerous, and form a cavernous network, the walls of which are firmly united to the tissue of the tumor, so that when divided or ruptured they are unable to retract. In such cases, injury to the tumor is often followed by profuse hemorrhage.

DEVELOPMENT.—The fibromata always originate from connective tissue, either from the cutis or subcutaneous connective tissue, from the submucous or subserous tissue, from fasciæ, the periosteum, the neurilemma, or from the connective tissue of organs. The process probably consists in the enlargement and multiplication of the connective-tissue cells. If this takes place very rapidly, the new growth presents at first the characters of embryonic tissue, the cells being very numerous, and the intercellular substance soft and amorphous (see "Development of New Formations"). The latter, however, subsequently fibrillates, the cells diminish in number and size, and the embryonic becomes fully developed connective tissue. The slower the process of development, the less marked are these embyronic characters. The fibromata are almost always limited by a fibrous capsule, which separates them from the surrounding structures. Their growth is slow and central, taking place within the capsule.

SECONDARY CHANGES.—Of these, partial mucoid softening and calcification are the most common. Ulceration also sometimes occurs in those growths which are situated in the submucous tissue.

VARIETIES.—Fibrous tumors present some variations in their characters, which depend for the most part upon the tissues from which they grow. Those growing from the cutis are softer and less dense in consistence than those met with in many other situations; they usually also have papillæ on their surface. These growths from the cutis are by no means uncommon, and may form enormous tumors. They are frequently multiple, and in their growth often become pedunculated. They commonly occur in middle or advanced life.

Another variety of fibrous tumor grows in connection with nerves; and is often described as *neuroma*. True neuromata, however—*i. e.*, new formations of nerve tissue—are amongst the rarest forms of new formations. These fibrous growths most frequently occur in connection with the superficial nerves. They grow from the neurilemma, and as they increase in size the nerve fibres become expanded over them, so that they often cause considerable pain; hence the term "painful subcutaneous tubercle," which is applied to them.

The fibroid tumors of the uterus, which are often regarded as fibrous tumors, appear in most cases to be overgrowths of the involuntary muscular tissue of the organ. They will, therefore, be described with the muscular tumors. (See " Myoma.")

PHYSICAL CHARACTERS, &c.—Fibrous tumors are usually more or less spherical or oval in shape, and are frequently lobulated on the surface. Their consistence varies; they may be exceedingly firm, dense, and fibrous, or softer and more succulent. On section, they usually present a grayish-white basis-substance, intersected with opaque, white glistening fibres. They are generally single, except when growing from the neurilemma or cutis, in which situations they are frequently multiple.

CLINICAL CHARACTERS.—Clinically the fibromata are perfectly innocent, and have little or no tendency to recur locally after removal.

CHAPTER XIII.

THE SARCOMATA.

THE Sarcomata are tumors consisting of embryonic connective tissue. Of these there are several varieties, depending upon the size and configuration of the cells, and the nature of the intercellular substance. They include what have generally been known in this country as *fibro-plastic*, *fibro-nucleated*, *recurrent fibroid*, and *myeloid* tumors.

Connective tissue in its embryonic condition is an immature tissue in a state of rapid development. It differs from the fully developed tissue in consisting almost entirely of cells, which are not only more numerous, but larger and rounder than those of the mature tissue. Its intercellular substance also, instead of being fibrous, is soft and amorphous, or only obscurely fibrillated. This is the common condition of connective tissue in the primary stages of all rapid formative processes, as already described when speaking of it as the tissue from which many tumors of the connective-tissue class originate (see "Development of New Formations").

In simple hyperplasias, as seen in the preceding chapter, the connective tissue may assume at first these embryonic characters; but in these, as in the embryo, it is ultimately developed into a mature structure—the cells diminish in number and size, and the intercellular substance fibrillates. In inflammatory conditions also of the connective tissue, the new growth differs in no way from embryonic tissue, but here cells of a lower type are subsequently formed, which undergo rapid retrogressive changes (see "Inflammation of Connective Tissue"). In the sarcomata, however, the connective tissue retains the embryonic state throughout its growth, there is a progressive formation of embryonic tissue; and thus the process differs from simple hyperplasia, on the one hand, in which a mature tissue is formed, and from the inflammatory one on the other, in which many of the new elements perish.

STRUCTURE.—The sarcomata may thus be defined as tumors consisting of connective tissue which throughout its growth retains the embryonic type. The *cells*, which constitute nearly the whole of the growth, consist for the most part of masses of nucleated protoplasm, and rarely possess a limiting membrane. They present many variations in size and form; as a rule, however, they preserve the same general characters in the same tumor. There are three principal varieties,—the *round*, the *fusiform*, and the *myeloid* cells.

The *round* cells are many of them indistinguishable from lymphatic cells or white blood-corpuscles. Others are larger and contain an indistinct nucleus with one or more bright nuclei; these closely resemble the granulation cells.

The *fusiform*, or *spindle-shaped* cells, are the so-called "fibroplastic cells." They are long narrow cells, terminating at each end in a fine prolongation. Some of them may be broader, approaching the epithelial type; others more or less stellate. They are slightly granular, and usually inclose a long oval nucleus, with or without nucleoli. In size they vary considerably. These cells represent a higher stage of development than the round cells, resembling those met with in granulation or embryonic tissue, which is in the process of forming mature connective tissue.

The *myeloid*, or *mother* cells, are much larger than either of the preceding, and are analogous to the cells met with in the medulla of bone in inflammatory conditions, and in the embryo. They are very irregular in shape, though for the most part more or less spherical, and they often possess numerous offshoots. They are finely granular, and contain several round or roundly-oval nuclei, each with one or more bright nucleoli. The nuclei may be exceedingly numerous, one cell containing as many as thirty. Both the cells and nuclei vary considerably in size.

An *intercellular substance* exists in all the sarcomata, although it is usually very small in quantity, the cells lying in nearly close apposition. It may be perfectly fluid and homogeneous, or firmer and granular, or, less frequently, more or less fibrillated. Chemically it yields albumen, gelatin, or mucin.

The *bloodvessels* are very numerous, and are either in direct contact with the cells, or separated from them by a little fibrillated tissue. Their distribution is very irregular, and their walls consist of embryonic tissue, similar to that of the growth which they supply; hence, the frequency with which rupture and extravasation of blood take place.

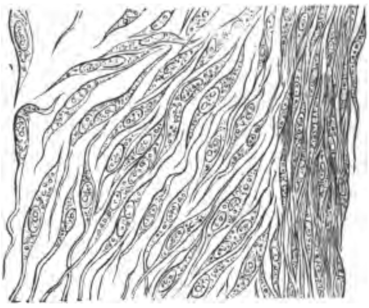
DEVELOPMENT.—The sarcomata always originate from connective tissue—either from the subcutaneous, the submucous, or the subserous tissues, the fasciæ, the connective tissue of organs, the peritoneum, or the medullary tissue of bones. Their growth may take place in two ways, by the multiplication of their own elements—*central* growth, and by the continuous invasion of their matrix—*peripheral* growth. A peripheral growth is the great characteristic of the sarcomata; they usually increase by the continuous invasion of their connective-tissue matrix, so that no line of demarcation exists between the two. Although the connective tissue alone constitutes the matrix from which they grow, they often appear, like the cancers, to implicate indiscriminately other tissues. This in reality is rarely the case; they usually merely displace these, and by their pressure cause them to atrophy. A purely central growth is less common. A sarcomatous tumor, however, frequently becomes encapsuled and growth takes place within the capsule; but even in this case the capsule is often merely that of the part within which the growth originates, as the periosteum, or the capsule of a lymphatic gland. It is probable that migrated white blood-corpuscles play an important part in the development of these tumors.

SECONDARY CHANGES.—The most important of these is fatty degeneration. This always occurs to a greater or less extent in the older portions of the growth, causing softening, caseation, or the production of cyst-like cavities. It is frequently associated with destruction of the bloodvessels and hemorrhage: the latter may give rise to pigmentation, and the formation of sanguineous cysts. Calcification, ossification, and mucoid degeneration are also common. The occurrence of ossification and pigmentation is influenced by the predisposition of the matrix from which the growth is produced: thus ossification is more prone to occur in tumors originating from the periosteum, pigmentation in those originating from the cutis or eyeball.

VARIETIES.—Although all the sarcomata possess the same general characters, they present many histological and clinical differences, which may serve as bases for their classification. The occurrence of the various secondary changes—ossification, pigmentation, mucoid degeneration, and the formation of cysts, impart their respective characters to the growth; hence *osteoid sarcoma*, *melanotic sarcoma*, and *cystic sarcoma*, have been described as distinct varieties. This is to a certain extent justifiable, inasmuch as sarcomata which have undergone these transformations, in many cases possess the property of reproducing the same characters when they occur secondarily in internal organs. The following histological classification, based upon the three different forms of cells already described, is perhaps the most convenient. It must, however, be borne in mind, that all the varieties of cells may be found in the same tumor, although the majority are usually of the same type; hence, the majority will determine the class to which the growth belongs.

1. *Spindle-celled Sarcoma.*—This, the well-known *fibro-plastic* tumor, is the most closely allied to the fibromas, inasmuch as it consists of a tissue which must be regarded as occupying an intermediate place between embryonic and fully developed connective tissue. It consists of spindle-shaped and fusiform cells, varying considerably in size, and nearly in close contact, there being very little intercellular substance. (Fig. 21.) The cells are parallel and arranged in bundles, which pass in all directions through the growth, so that on section it presents a somewhat fasciculated appearance. In some parts there may be partial fibrillation. The spindle-celled sarcomata grow from the periosteum, the fasciæ, and from the connective tissue in other parts.

FIG. 21.

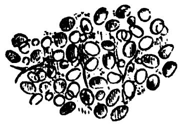


Spindle-celled Sarcoma. × 350. (Virchow.)

They are more frequently encapsuled than the other varieties; they nevertheless very frequently extend by peripheral growth.

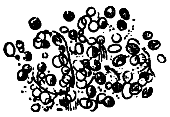
2. *Round-celled Sarcoma.*—This is of softer consistence than the preceding, and from its resemblance in many cases to encephaloid, has received the names of *medullary*, *encephaloid*, or *soft* sarcoma. Histologically it is elementary embryonic tissue, consisting of the round cells already described, imbedded in a scanty, and usually soft, homogeneous, or finely granular intercellular substance. (Fig. 22.) There is an almost complete absence of fusiform cells, and of the partial fibrillation which is so frequent in the more highly developed spindle-celled variety. The round-celled sarcomata are of a uniform soft brain-like consistence, and of a somewhat translucent grayish or reddish-white color. On scraping the cut surface, they yield a juice which is rich in cells. They are exceedingly vascular, the vessels often being dilated and varicose, and from their liability to rupture they frequently give rise to ecchymoses, and to the formation of sanguineous cysts. They grow from the cutis, the subcutaneous cellular tissue, the periosteum, the fasciæ, and from the connective tissue of organs. They extend rapidly by peripheral growth, and are rarely encapsuled. From their clinical and physical characters, these tumors are very liable to be confounded with encephaloid cancer: they are distinguished by the absence of a fibrous stroma, by the uniformity in the character of their cells, and by the absence of any invasion of the surrounding structures in their growth other than the connective tissue from which they grow.

FIG. 22.



Round-celled Sarcoma. × 200.

FIG. 23.



Glioma. × 200.

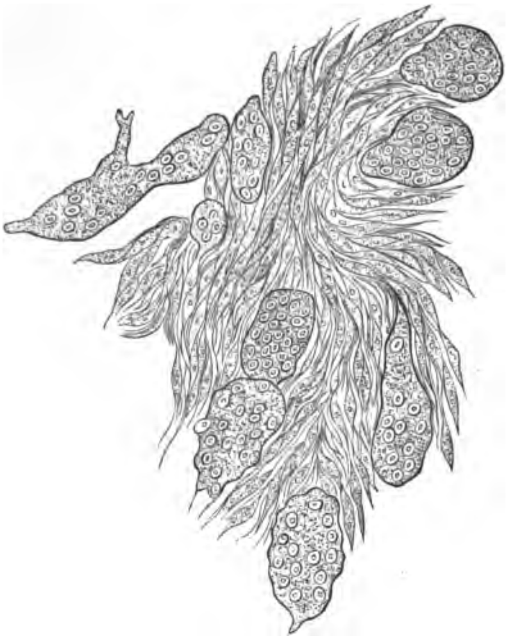
There is a variety of round-celled sarcoma growing from the connective tissue of nerve, the "neuroglia," known as *Glioma.* It consists of small round cells, imbedded in a scanty, homogeneous, granular, or slightly fibrillated intercellular substance. (Fig. 23.) Some of the cells may possess fine prolongations which, by communicating with one another, form a reticulated structure. These tumors are not encapsuled; they occur in the gray and white substance of the brain, in the cranial nerves, and in the retina. They never cause secondary growths. Childhood is the age at which they are most frequently met with.

3. *Myeloid Sarcoma.*—This, which is commonly known as *myeloid*, is closely allied to the preceding variety, consisting of elementary embryonic tissue. It possesses, however, certain histological peculiarities depending upon the characters of the tissue from which it grows. Myeloid tumors always occur in connection with bone, and almost invariably originate in the medullary cavity. They consist of the large, many-nucleated cells already described as "myeloid cells,"—which are merely the cells of the medulla in a state of excessive nutritive activity—together with numerous fusiform cells like those met with in the spindle-celled varieties. There are also some smaller cells resembling those of the medulla in its normal condition. These various forms of cells are nearly in close contact, there being very little intercellular substance. (Fig. 24.) The growth is usually exceedingly vascular.

Myeloid tumors always grow in connection with bone, the heads of the long bones being their favorite seat. They are also frequently met with in the upper and lower jaws (Epulis). As they increase in size, the compact tissue of the bone becomes expanded over them, and they thus often communicate on palpation the peculiar sensation of "egg-shell crackling." In many cases their great vascularity gives rise to distinct pulsation, and to the generation of an aneurismal murmur: this is most frequent in those of the lower extremity. These tumors are of moderately firm consistence, many of them are firm and fleshy, others are softer, more resembling size-gelatin. They are not pulpy and grumous like the soft sarcomas, neither do they present the fasciculated appearance of the spindle-celled varieties. Their cut surface has a uniform succulent appearance, often mottled with patches of red. They are usually encapsuled by the periosteal covering of the bone within which they grow.

Clinical Characters.—The sarcomata occur most frequently in early and middle life, and are amongst the most malignant of new formations. They are characterized by their great tendency to extend locally and to recur after removal, and by their power of reproducing themselves in internal organs. They rarely infect the lymphatic glands. Their growth is in many cases exceedingly rapid, and the softer forms usually increase by the continuous invasion of their matrix: they rarely, however, like cancer, implicate other tissues. Their characteristic clinical feature is their tendency to become generally disseminated, the secondary growths occurring for the most part in the lungs. The dissemination is effected by means of the blood, and this is owing to the thinness of the walls of their bloodvessels and to the immediate contact of these with the cells of the growth—conditions most favorable to the entrance of the cellular elements into the circulation. The dissemination of the sarcomata, is, on this account, often more rapid than that of the cancers. In the latter, extension in the early stage, takes place principally by the lymphatics, and dissemination by the blood only occurs in the later stages of the disease. The secondary sarcomata frequently resemble the primary one, in other cases the several varieties replace one another.

FIG. 24.



Myeloid Sarcoma. (Virchow.)

These malignant characters are possessed by the different varieties of sarcoma, in different degrees. As a rule, the softer and more vascular the tumor, and the less its tendency to form a fully developed tissue, the greater is its malignancy. The soft, round-celled varieties, are thus usually much more malignant than the firmer spindle-celled growths. This is probably partly owing to the small round cells being endowed with greater powers of spontaneous movement than the spindle-shaped and larger cells, hence they more readily make their way into the bloodvessels. The myeloid growths are perhaps the least malignant; they may however also give rise to secondary growths in internal organs.

CHAPTER XIV.

THE GUMMATA.

THE Gummata are new formations consisting of a very incompletely organized granulation tissue. The primary result of all active formative processes on the part of the connective tissue, is, as already described in the preceding chapter, and in that on the general pathology of the new formations—the formation of an embryonic tissue. The more active the process, the more elementary is this new tissue, and the more closely does it resemble that met with in the inflammatory process, which is known as *granulation tissue.* (See " Inflammation of Connective Tissue.")

Granulation tissue must thus be regarded as the most elementary form of embryonic tissue. It is met with not only in an ordinary granulation, but also in the rapidly growing connective tissue of the embryo, and as the primary stage of all rapidly developed new formations which originate from connective tissue. Structurally, it consists of small, round, finely granular cells from $\frac{1}{1800}$ to $\frac{1}{2500}$ inch in diameter, inclosing an ill-defined nucleus, which is often only visible after the addition of acetic acid. These cells possess no limiting membrane, and are imbedded in a very scanty, soft, homogeneous intercellular substance. (See Fig. 19.)

There is a certain class of new formations which have been described by Virchow as "granulation tumors." They consist in the first place of a granulation tissue; this becomes very incompletely organized into a fibrous structure, and many of the elements at the same time rapidly undergo retrogressive changes, so that the growth is ultimately made up of atrophied, degenerated, and broken down cell-products, imbedded in an incompletely fibrillated tissue. These are the growths which are most characteristic of syphilis, and they are known as *gummata* or *gummy tumors.* They are closely allied to chronic inflammatory growths on the one hand, and to the small round-celled sarcomata on the other.

Although the gummata are the new formations most characteristic of syphilis, all syphilitic growths cannot be included under this head. A simple fibrous growth is as frequently the result of the syphilitic poison as a true gummy tumor, although as evidence of syphilis it occupies an inferior place. The two, however, are so frequently associated, that in many cases it becomes difficult to draw a sharp line of demarcation between them; a formation which in its early stage is simply young connective tissue, may subsequently assume either in whole, or in part, the characters of a gummy tumor. It will therefore be well before describing the gummata to say a few words on the simple fibrous growths.

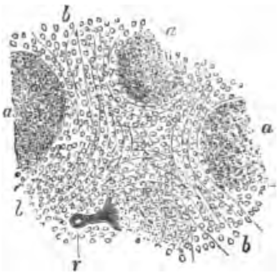
New growths of connective tissue occur for the most part earlier in the course of constitutional syphilis, than the gummy tumors. They are the homologous growths of syphilis, whereas the gummata are more heterologous. They are met with in many situations. In the *periosteum*, where they constitute "nodes," they are situated in the deeper layers of the membrane, which proliferate and form an embryonic tissue (periostitis). This may develop into a fibrous structure, or it may become a true gummy growth. These changes are usually attended with thickening of the subjacent bone.

In the *liver*, an increase of fibrous tissue is very common as the result of syphilis;—both the capsule and the interlobular tissue may be involved. The affection of the capsule consists in an irregular thickening and puckering of its substance; the thickened portions being frequently connected with dense fibrous septa, which pass into the interior of the organ. There are usually also numerous strong peritoneal adhesions. The interlobular change is very similar to that of ordinary cirrhosis. It differs in being less uniform and general, the increase of connective tissue occurring only in certain regions, or around certain branches of the portal vein; also in the intercellular network being much more extensively involved, the new growth often extending to the centre of the hepatic lobules. The organ thus becomes much more irregularly puckered and atrophied than in non-syphilitic cirrhosis. True gummy growths may originate subsequently in the fibrous septa.

In the *lungs*, *testicle*, *muscle*, and in other parts, a similar hyperplasia of the connective tissue may take place, although it is less frequent in these situations than in the liver and periosteum. In all, however, the new tissue is characterized by the irregularity of its distribution, and by its tendency to become subsequently the seat of gummy growths.

STRUCTURE OF GUMMATA.—The Gummata consist of atrophied and degenerated elements imbedded in a scanty and obscurely fibrillated stroma. The central portions of the growth are composed almost entirely of closely packed granular débris, fat-granules, and cholesterine, amongst which there may be an exceedingly scanty fibrillated tissue. Surrounding this and directly continuous with it, is a more completely fibrillated structure; whilst the peripheral portions of the growth—which are continuous with the surrounding tissue—consist entirely of small round cells, resembling granulation-cells and lymph-corpuscles. (Fig. 25.) The bloodvessels, which only exist in the external portions of the growth, are very few in number.

FIG. 25.



Gummy Growth from Liver. *a.* Central portions of growth, consisting of granular débris. *b.* Peripheral granulation tissue. *r.* A bloodvessel. × 100. (Cornil and Ranvier.)

DEVELOPMENT.—The gummata always originate from connective tissue. The first stage in the process consists in the formation of a granulation tissue. This is followed by the incomplete development of the new tissue into a fibrous structure, together with the atrophy and degeneration of many of the young elements. The degenerated elements become closely packed in the centre of the growth, whilst proliferation and incomplete fibrillation continue at the circumference. The three zones above described, thus correspond with the three different stages of the growth;—the peripheral zone, which is continuous with the surrounding tissue, is the earliest stage, that of cell-proliferation; the central zone represents the oldest portions of the growth, which have undergone retrogressive changes, whilst the intermediate and more fibrous zone is the most perfectly developed tissue. The bloodvessels, which only exist in the zone of proliferation, appear to become obliterated in the process of development, and this probably accounts for the rapid degeneration of the central portions of the growth. When the tumor is large, it may sometimes be seen during the period of its development, to be made up of several distinct smaller growths, each presenting at its circumference the more perfect cells, whilst its central parts are granular and amorphous.

SECONDARY CHANGES.—The only secondary change to which the gummata are liable, is the gradual drying up of the growth, and its transformation into a firm and somewhat caseous mass. This change is characteristic of these tumors, and is one which they invariably undergo; and upon it their peculiar physical characters depend. In some cases the process of absorption may be carried still further, and nearly the whole of the mass become absorbed, leaving merely the fibrous stroma in the form of a cicatrix. If the growth is situated in the submucous tissue, the mucous membrane may become destroyed, and an ulcer form. This is seen in the pharynx, soft palate, and other parts.

PHYSICAL CHARACTERS, &c.—The physical characters of the gummata are such as will have been already inferred from the description of their structure. During the earliest stages of their development, when they but rarely come under observation, they are growths of a soft fibrous consistence. As usually met with, however, when their growth is more complete, they are firm yellowish-white masses, having in many situations the appearance, on section, of a horse-chestnut. These are surrounded by a translucent fibrous-looking tissue, which often has somewhat the appearance of a capsule. They vary considerably in size, and from their mode of growth, are associated in such a way with the surrounding structures that their limits are very imperfectly defined, and their complete enucleation is impossible.

The gummata are the new formations most characteristic of syphilis, and their distribution is almost coextensive with that of syphilitic lesions. They are met with in the skin and subcutaneous cellular tissue, in the submucous tissue, in intermuscular septa and fibrous membranes, in bones, and in the connective tissue of organs—especially of the liver, brain, testicle, and kidney. Some forms of periosteal node, the deep ulcerating growths in the pharynx, soft palate, tongue, larynx, and in other parts, are all examples of the same formation.[1]

The simple fibrous growths, as already stated, often subsequently become gummy tumors: this is seen in the growths beneath the periosteum (nodes), in the liver, and in other parts.

Clinical Characters.—The existence of gummy tumors must be regarded as the strongest evidence of the existence of constitutional syphilis, especially if they are multiple, and generally distributed. These growths do not possess the property of reproducing themselves in other parts, they are, therefore, clinically, innocent.

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[1] These true gummy ulcerations must be distinguished from the *superficial* ulcerations resulting from hyperplasia of the lymphatic structures, which also occur in syphilis.

CHAPTER XV.

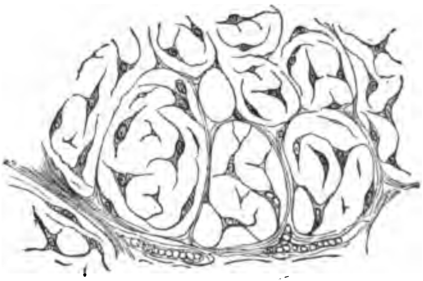
THE MYXOMATA.

THE Myxomata are tumors consisting of mucous tissue. Mucous tissue is a translucent and succulent connective tissue, the intercellular substance of which yields mucin. Physiologically, this tissue is met with in two forms, and in two situations: one—in the vitreous humor of the eye, in which the cells are roundish and isolated, the other—in the umbilical cord, in which the cells are fusiform or stellate, and give off fine trabeculæ which anastomose with one another. In both, the intercellular substance is homogeneous and yields mucin. The connective tissues generally in their embryonic condition, as already stated when describing "mucoid degeneration," possess an intercellular substance containing large quantities of mucin. New formations may undergo a mucoid change, and thus closely resemble in their physical and chemical characters, the myxomas. A myxoma, however, is a growth which from its commencement consists of mucous tissue. The myxomata are thus very closely allied to the sarcomata, and by many are included in the same class of new formations.

STRUCTURE.—The cells present the two varieties met with in the physiological tissues. The majority are angular and stellate, with long anastomosing prolongations and trabeculæ. (Fig. 26.) Others are isolated, and fusiform, oval, or spherical in shape. They usually possess one, in some cases two distinct nuclei. Their contour is very indistinct, owing to the refracting nature of the intercellular substance. The latter is very abundant, perfectly homogeneous, of a soft gelatiniform consistence, and yields large quantities of mucin; amongst it are a varying number of amœboid cells The bloodvessels, which are not numerous, are readily visible and easily isolated. A few elastic fibres are sometimes seen between the cells.

DEVELOPMENT.—The myxomata always originate from one of the connective tissues. Adipose tissue is their most favorite seat—either the subcutaneous, the submucous, or the intermuscular adipose tissue. They also grow from the medullary

FIG. 26.



Myxoma. × 300. (Rindfleisch.)

tissue of bone, the connective tissue of organs, and from the connective tissue of the brain, spinal cord, and nerves. They are usually separated from the surrounding structures by a fibrous capsule, although they sometimes increase by the continuous invasion of their matrix. Their growth is usually slow, but they may attain an enormous size.

SECONDARY CHANGES.—Of these the most common is rupture of the capillaries, hemorrhage, and the formation of sanguineous cysts; this, however, is less frequent than in the sarcomata. The cells themselves may undergo mucoid or fatty degeneration, and thus be destroyed; this is usually accompanied by liquefaction of the intercellular substance.

VARIETIES.—The varieties of myxoma depend principally upon its combination with other growths. The most common is a combination with lipoma, adipose tissue being the tissue from which it most frequently originates. Combinations with sarcoma and enchondroma are also frequently met with.

PHYSICAL CHARACTERS, &c.—The myxomata are of a peculiar, soft, gelatiniform consistence, and of a pale grayish or reddish-white color. On scraping the cut surface, they yield a tenacious, mucilaginous liquid, in which may be seen the cellular elements of the growth. They are most frequently met with in the later periods of life. Their most common seats are those of adipose and nervous tissue. They may also grow from the placenta, constituting the so-called "hydatiform degeneration." When situated in superficial parts they may become pedunculated. In the submucous tissue of the nose, they constitute one form of nasal polypus.

Clinical Characters.—Clinically, the myxomata are for the most part benign growths. If completely removed, they rarely recur. Sometimes, however, they exhibit malignant characters, recurring both locally and in internal organs. Probably, in many cases, these malignant characters are owing to the combination of the myxoma with some other growth.

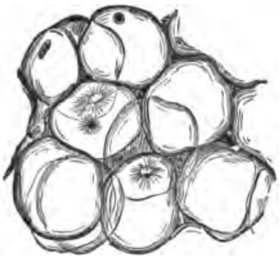
CHAPTER XVI.

THE LIPOMATA.

A GENERAL new formation of adipose tissue constituting *obesity*, has already been described under "fatty infiltration." A localized and circumscribed formation, constitutes a *lipoma* or fatty tumor.

STRUCTURE.—The lipomata resemble in their structure adipose tissue. (Fig. 27.) They consist of cells containing fat, and a variable quantity of common connective tissue. The cells, like those of adipose tissue, though usually somewhat larger—are more or less round, or polygonal in shape, and are distended with fluid fat. The nucleus and protoplasm are so compressed against the cell-wall by the fluid contents, that although their existence may always be demonstrated by treatment with reagents, they are usually only readily visible when the cell is atrophied and contains less fat. (See Fig. 2, *a*.) The connective tissue, which varies in amount, unites the cells in masses or lobules of various sizes, and also in most cases, forms a capsule around the tumor. Bloodvessels are distributed in the fibrous septa.

FIG. 27.



Lipoma. Some of the cells contain crystallized fatty acids. × 200.

DEVELOPMENT.—The lipomata grow from adipose, or from common connective tissue. Adipose tissue, it must be remembered, is merely connective tissue containing numerous cells which are infiltrated with fat; and its growth consists, either in the infiltration of more of these cells, or in a proliferation of the cells, and an accumulation of fat in those newly developed. A lipoma in the same way originates by a localized proliferation of cells, which as they are produced become infiltrated with fat. The growth of these tumors is always very slow, and they are usually encapsuled by a layer of fibrous tissue.

SECONDARY CHANGES.—Secondary changes in the lipomata are not common—their fibrous septa may, however, become calcified, or even ossified, and the fatty tissue undergo a process of liquefaction. Softening may also occur from a mucoid change. Sometimes they become the seat of an inflammatory process, then when situated in the subcutaneous tissue, the skin over them becomes adherent and ulcerates, and a fungating mass is the result.

PHYSICAL CHARACTERS, &c.—The situation of the lipomata is almost coextensive with that of adipose and connective tissue. They occur most frequently, however, in those parts in which fat is normally met with, as in the subcutaneous tissue, the intermuscular septa, and in the connective tissue of glands. They also occur in the subsynovial tissue, and in the submucous tissue of the stomach and intestines. They sometimes attain an enormous size. They are lobulated, and are usually surrounded by a fibrous capsule which separates them from the adjacent structures. On section they present the ordinary appearance of adipose tissue. Their consistence varies with the amount of fibrous tissue which they contain. They are usually single, though not unfrequently multiple. In their growth they sometimes become pedunculated.

CLINICAL CHARACTERS.—Clinically, the lipomata are perfectly innocent.

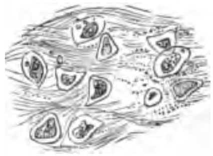
CHAPTER XVII.

THE ENCHONDROMATA.

The Enchondromata are tumors histologically resembling cartilage.

Structure.—Like cartilage they consist of cells and an intercellular substance, which present all the variations observed in the normal tissue. (Fig. 28.) The intercellular substance may be hyaline, faintly or distinctly fibrous, or mucoid. When fibrous, the fibres may be arranged like those of fibrous cartilage, or more or less concentrically around the cells as in the reticular cartilages of the ear and larynx. The cells may be very numerous, or few in proportion to the matrix. They are round, oval, spindle-shaped or stellate. In the hyaline forms they are usually large and round or oval; in the fibrous forms they are often smaller and spindle-shaped, more resembling those of connective tissue; and in the mucoid forms, they are more commonly stellate and branched, like those of the umbilical cord. They are either single or arranged in groups, and are usually surrounded by a fibrous capsule, though this is often very indistinct. They inclose one or more nuclei and slightly granular contents; sometimes a cell-wall cannot be distinguished. In addition to the intercellular substance, the growth is usually divided into several lobes, by bands of fibrous tissue; these lobes are often very distinct, so that the growth appears to be made up of several separate tumors. The fibrous tissue in most cases also encapsules the growth and separates it from the surrounding structures. The vessels, which are often very numerous, are distributed in the fibrous septa.

Fig. 28.



Enchondroma. × 200.

Development.—The enchondromata most frequently originate from bone and common connective tissue, very rarely from cartilage. Cartilage itself, and especially fibrous cartilage, is very closely allied to connective tissue; it is developed from connective tissue, and never from cartilage or bone. It grows from the deeper layers of the perichondrium, which proliferate and form an embryonic tissue; the young cells become cartilage-cells, and these probably form the matrix, which is either homogeneous or fibrillated, constituting in the one case hyaline, and in the other fibrous cartilage. The development of enchondroma from connective tissue is precisely similar to the physiological process.

In the development of enchondroma from osseous tissue, the medulla is the source of the new growth. This proliferates, the osseous trabeculæ are absorbed, the neighboring medullary spaces open one into the other, and in this manner a large medullary cavity is produced. In the centre of this, the young cells first formed, enlarge and become separated by a homogeneous, or less frequently, slightly fibrillated, intercellular substance, and thus is produced a mass of cartilage in the centre of the medullary tissue. This gradually increases till ultimately a layer of fibrous tissue is formed around it, and its further growth takes place from the tissue of its capsule.

Lastly, cartilaginous growths may originate from cartilage itself. These are seen on the surface of the articular cartilages in chronic rheumatism, and sometimes on the costal and intervertebral cartilages. They are simply local outgrowths, from pre-existing cartilage. They rarely attain a large size, and in structure and physical characters, more closely resemble normal cartilage than the other forms of enchondroma. They are often described as *enchondroses.*

SECONDARY CHANGES.—Of these, calcification is perhaps the most common. It affects different parts of the growth, commencing in the capsules, and then involving the intercellular substance. Ossification also frequently occurs; it commences at separate centres, and spiculæ of bone are formed, which traverse the tumor in various directions. Fatty degeneration and mucoid softening are common changes, and may lead to the formation of large softened masses which present the appearance of cysts. In rare cases the skin covering the tumor ulcerates, and a fungating mass protrudes.

VARIETIES.—The varieties of enchondroma depend upon the nature of the intercellular substance. There are thus hyaline,

[illegible]

closely allied to connective tissue; it is developed from connective tissue, and never from cartilage or bone. It grows from the deeper layers of the perichondrium, which proliferate and form an embryonic tissue; the young cells become cartilage-cells, and these probably form the matrix, which is either homogeneous or fibrillated, constituting in the one case hyaline, and in the other fibrous cartilage. The development of enchondroma from connective tissue is precisely similar to the physiological process.

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VARIETIES.—The varieties of enchondroma depend upon the nature of the intercellular substance. There are thus hyaline, fibrous, and mucoid enchondromata; these, however, are usually combined in various degrees in the same tumor. As a rule, those originating from the medulla of bone, are of the hyaline and mucoid class, whilst those originating from connective tissue in other situations, are more frequently fibrous. The rapidly growing fibrous forms approach very closely the confines of the sarcomata, the mucoid forms the confines of the myxomata; and these varieties often appear to be associated in the same tumor. Enchondroma is sometimes associated with encephaloid cancer, especially in the testicle.

A variety of enchondroma has been described under the name of *osteo-chondroma*, which in structure more closely resembles bone than cartilage. It consists of a tissue similar to that met with between the periosteum and bone in rickets, which from its resemblance to osseous has been called *osteoid* tissue. This tissue only requires calcifying to become true bone. Like bone it is made up of trabeculæ and medullary spaces, but the trabeculæ, instead of bone-corpuscles and lamellæ, consist of small angular cells without a capsule, situated in an obscurely fibrillated matrix, which in part is calcified. The medullary spaces contain a fibrous stroma and many bloodvessels. The osteo-chondromata, although consisting mainly of this osteoid tissue, contain also a small proportion of cartilage. They originate beneath the periosteum, their common seat being the ends of the long bones. Their growth is very rapid, and they often attain an enormous size.

PHYSICAL CHARACTERS, &c.—The enchondromata occur most frequently in early life. About three-fourths of them are met with in the osseous system, where they grow either from the medulla or from the periosteum: their favorite seat is the extremities of the fingers and toes. The remaining fourth occur most frequently in the parotid gland and in the testicle. They occasionally grow in the subcutaneous cellular tissue of the mammæ and lungs. They are usually single except when occurring on the fingers and toes, in which situations they are more frequently multiple. They consist of a single tumor, or of several smaller tumors held together by fibrous tissue. Their consistence is softer than that of cartilage, sometimes it approaches that of a soft jelly. Their growth is usually slow, though in the softer forms and in those growing in the medulla of bone, it is often very rapid. They may attain an enormous size.

Clinical Characters.—The enchondromata must for the most part be regarded as innocent growths. Those homologous forms which originate from cartilage, and have been called "Enchondroses," differ in all respects from the heterologous, and never exhibit malignant characters. The more heterologous forms originating from connective tissue and bone, are usually encapsuled, and in most cases produce merely local effects, although these, from the parts involved and the rapidity of growth, are often very injurious. The softer forms, however, and especially those which occur in the medulla of bone, sometimes exhibit malignant characters. These grow the most rapidly, and are often not limited by a fibrous capsule; they may therefore recur locally after removal. In some cases they have also infected the lymphatic glands, and recurred in the lungs.

CHAPTER XVIII.

THE OSTEOMATA.

THE Osteomata or osseous tumors are tumors consisting of osseous tissue. A new formation of bone occurs under various circumstances. Irritative conditions of the bone and periosteum are often attended by a large formation of new bone. This is seen after fractures, in which there is not only a formation of bone from the bone itself, but also from the periosteum and adjacent fibrous structures (permanent and provisional callus). Chronic inflammation of the periosteum is also frequently followed by thickening of the bone beneath it. These, however, are inflammatory formations, and have not an independent growth like the osseous tumors.

STRUCTURE.—Osseous tumors in structure resemble normal bone. There are three varieties:

1. *The Eburnated Osteomata.*—These consist of dense, compact, osseous tissue. The lamellæ are arranged concentrically and parallel to the surface of the tumor. There is a complete absence both of bloodvessels and of cancellous tissue.

2. *The Compact Osteomata.*—These are formed of a tissue similar to that of the compact tissue of the long bones; differing only in the arrangement of the Haversian canals and canaliculi, which is less regular than in normal bone.

3. *The Cancellous Osteomata.*—These consist of cancellous osseous tissue. The medullary spaces may contain embryonic tissue, a fibrillated tissue, or fat.

DEVELOPMENT.—Osseous tumors like normal bone, can originate only from cartilage or from connective tissue. In both cases the process is precisely similar to that of physiological ossification.

VARIETIES.—The osteomata are divisible into two classes, according to their seat,—the homologous osteomata, or *exostoses*, and the heterologous osteomata, or *osteophytes*.

The *homologous osteomata* or *exostoses* are outgrowths from proexisting bone, growing either from the periosteum, from the articular cartilage, or from the medulla. Those growing from the periosteum occur most frequently on the external and internal surfaces of the skull; the orbit is an especially favorite seat, and here they are often dense and eburnated. They are also common on the upper and lower jaws, and in this situation they may grow from the dental periosteum. There is usually a line of demarcation between them and the subjacent bone, the new tissue of the tumor being distinct from the compact tissue of the bone. The periosteum from which they grow covers them, and is continuous with that of the old bone.

The exostoses growing from the articular cartilages occur at the ends of the long bones. In structure they are much more cancellous than the periosteal growths, and their outline is less regular. The medullary exostoses—or more properly, *en-ostoses* are the least frequent; they originate in the medullary tissue.

The *heterologous osteomata* or *osteophytes* originate apart from bone, growing from the connective tissue, or from cartilage. They are much less common than the homologous growths, and must, in most cases, be regarded rather as inflammatory formations than as tumors. Such formations of bone are met with in tendons, in the cartilages of the larynx in chronic laryngitis, in the bronchi, in articular synovial membranes, in muscle, in the arachnoid and pia mater, and occasionally in the lungs and brain. They must be distinguished from calcareous deposits, in which there is no new formation. (See "Calcareous Degeneration.")

CLINICAL CHARACTERS.—The osteomata are perfectly innocent tumors. Their growth is very slow, although they sometimes attain a considerable size. They are often hereditary and multiple, in which case they usually occur in early life. Those osseous growths which sometimes exhibit malignant characters, are sarcomata, enchondromata, or cancers which have undergone partial ossification. From these, true osteomata must be carefully distinguished.

CHAPTER XIX.

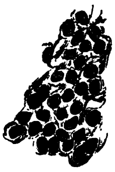
THE LYMPHOMATA.

THE Lymphomata are new formations, consisting of lymphatic, or, as it is more commonly called, *adenoid* tissue. Adenoid tissue is the tissue composing the follicles of the lymphatic glands, and the Malpighian corpuscles of the spleen, and existing in many other organs belonging to the lymphatic system. This tissue is now known to have a much more general distribution than was formerly supposed; it not only constitutes the follicles of the lymphatic glands, and the Malpighian corpuscles of the spleen, but also Peyer's glands, and the solitary glands of the intestine, the follicles of the pharynx and tonsils, the Thymus gland, and the trachoma glands of the conjunctiva. Recently it has also been found to exist around the bloodvessels of the pia mater and of other parts, in the peribronchial connective tissue, in the pleura immediately beneath its epithelium, in the peritoneum, in the mucous membrane of the alimentary canal, and in the medulla of bone.

STRUCTURE.—Adenoid tissue, wherever it exists, possesses the same general structure, and the follicle of a lymphatic gland may be taken as the type, not only of the physiological tissue, but also of the pathological growths.

This tissue consists essentially of a delicate reticulum, within the meshes of which are contained lymphatic cells—the so-called lymph-corpuscles. The reticulum is made up of very fine fibrils, which form a close network, the meshes of which are only sufficiently large to inclose a few, or even a single corpuscle, in each. (Fig. 29.) The fibrils usually present a more or less homogeneous appearance, and amongst them there are a few scattered nuclei.

FIG. 29.



Adenoid Tissue from Lung, in a case of Chronic Phthisis. × 400.

The lymphatic cells, or lymph-corpuscles, which constitute the greatest part of the tissue, can in most cases be readily isolated from the meshes of the reticulum. They are identical in their characters with the white corpuscles of the blood, and like these are contractile, and possess the power of spontaneous movement. As usually seen after death, they are spheroidal, pale, semi-transparent bodies, varying considerably in size, and also presenting slight differences in their structure. Some are granular, and appear to possess no nucleus; in others, a distinct, simple, or compound nucleus is visible, which is usually also granular; others again are much larger, and contain two or even three nuclei. (See Fig. 31.)

DEVELOPMENT.—The lymphomata originate for the most part from adenoid tissue, being simply overgrowths of pre-existing lymphatic structures. At the same time it is probable that the new elements are in some cases partly derived from migrated blood-corpuscles, which multiply subsequently to their escape from the vessels. (See "Leukæmia.") These growths are therefore usually *homologous*. They may, however, be in a certain sense *heterologous*, either owing to the new tissue extending considerably beyond the confines of the old, or to its growth in situations where it is normally almost entirely wanting. Whether they are ever heterologous in the strict sense of that term, originating from any other than lymphatic tissue, appears to be extremely doubtful.

VARIETIES.—The lymphomata include the three following varieties: *simple lymphoma*, *leukæmia*, and *tubercle*. Each of these must be considered separately.

SIMPLE LYMPHOMA.

A simple non-inflammatory enlargement of a lymphatic gland, not resulting from any obvious irritation, undergoing no retrogressive changes, and like a tumor having a tendency continually to increase, may be called a *simple lymphoma*.

The enlargement is owing to a hyperplasia of the elements of the gland—a hyperplasia which is characterized by its *chronicity*, and *tendency to continue*. The newly-formed elements undergo no retrogressive changes, and hence the size of the gland becomes permanently increased. The lymph-corpuscles throughout the gland increase in number, many of them at the same time increase in size, and the larger cells often contain several nuclei. Whether the migration of blood-corpuscles takes any part in the process, is unknown. If this general hyperplasia is rapid, the gland becomes soft and pulpy in consistence, and the cortical and medullary portions are no longer distinguishable. As the growth continues, there is usually at the same time an increase in the stroma of the gland, the trabeculæ and capsule become denser and more fibrous, so that ultimately the consistence of the enlarged gland may be firmer than natural. The slower and more chronic the growth, the greater is the increase in the fibrous structures.

Sometimes the enlargement of the glands appears in the first place to be of an inflammatory nature, and to result from some irritation, but upon this being removed, the glands instead of subsiding continue to increase. In most cases, however, no such source of irritation is discoverable.

The glands which are especially prone to this disease are the cervical, the submaxillary, the axillary, the inguinal, the bronchial, and the abdominal glands. Usually only a single gland or a single group of glands is affected; sometimes, however, the growth is more general. As the glands enlarge, they gradually unite, so that ultimately they may form very large lobulated tumors. If these are situated in the thorax, they may by their pressure cause death.

The lymphatic structures in the intestine may in the same way become enlarged, and project so as to form polypi. The enlargement of the spleen in ague is also probably of the same nature.

LEUKÆMIA.

Leukæmia is a disease characterized by a permanent and continuous increase in the number of the white corpuscles in the blood, which increase is associated with a new formation of lymphatic tissue in the spleen, in the lymphatic glands, and sometimes in other organs. Respecting the relation which subsists between the new growth of lymphatic tissue and the increase in the number of the white blood-corpuscles, it is probable, as stated by Virchow, that the former is a cause of the latter, and that the increase is partly due to a larger number of lymphatic elements entering the blood than in health. At the same time this cannot be admitted to be the only cause of the increase, as new growths of lymphatic tissue may take place in these organs without the production of any leukæmia. It is probable that the power of the white blood-corpuscles and lymph-corpuscles to form red corpuscles is diminished. Possibly, also, the white corpuscles may increase by multiplication in the blood.

The new formation of lymphatic tissue is in the first place *hyperplastic*, taking place in the spleen and in the lymphatic glands; subsequently, however, it may become *heteroplastic*, and the liver, the kidneys, the lungs, and other organs, may become infiltrated with lymphatic elements. The growth is a *continuous* one; the new elements, many of them, enter the blood, the remainder, not undergoing any retrogressive changes, give rise to an increase in the size of the organs in which they are generated.

The new growth may commence in the spleen or in the lymphatic glands. In most cases the spleen alone is involved. Sometimes the growth in the spleen is associated with a similar one in the lymphatic glands. In rare cases, the lymphatic glands are the sole seats of the hyperplastic process.

In the *spleen*—which must thus be regarded as the most important organ in the production of leukæmia—the first stage in the process is one of hyperæmia. The organ becomes exceedingly vascular, and the cells, both of the pulp and of the Malpighian corpuscles, increase in number. The process, however, soon becomes principally limited to the Malpighian corpuscles. In these the cells continue to multiply rapidly, many new blood-vessels are formed, and the corpuscles thus increase in size. They sometimes attain the size of a hazel-nut, and are seen as firm, whitish nodules of an irregular shape, scattered through the much enlarged and vascular organ. As the Malpighian corpuscles increase in size, the surrounding pulp gradually atrophies, and often becomes deeply pigmented. The trabeculæ at the same time become thicker and more fibrous. The organ thus becomes firmer in consistence, and the enlarged Malpighian corpuscles make up the chief part of its bulk. The capsule also becomes thickened, and forms adhesions with the surrounding viscera.

In the *lymphatic glands* the process is precisely similar to that in the spleen, the follicles of the gland being the chief seats of the hyperplasia. The glands become enlarged, soft, and vascular.

In course of time the disease may become *heteroplastic*, and a large formation of lymphatic tissue take place in organs, in which normally it is almost entirely wanting. This heteroplastic tissue consists for the most part of white blood-corpuscles, which have migrated from the enlarged and dilated vessels, and have infiltrated the surrounding structures; it must, therefore, be regarded as an *infiltration* rather than as a true new formation. It is probable, however, that it partly originates either from the connective tissue of the organ, or from the perivascular adenoid tissue.

The organ which is the most frequent seat of the heteroplastic growth is the *liver*. Here, the vessels throughout the organ become enlarged and distended with white blood, and the infiltration commencing in the interlobular tissue, gradually extends into the lobules themselves. This is, in most cases, attended by an increase in the size of the hepatic cells. The liver thus becomes considerably enlarged, the lobules are large and distinct, and are in many parts seen to be separated by a grayish-white substance, which is the new lymphatic tissue. As the infiltration extends the lobules become compressed and atrophy, and the new tissue is seen as grayish-white masses scattered through the substance of the organ. Associated with this infiltration there is often a formation of small round nodules, closely resembling miliary tubercle, from which they are distinguished, however, by the absence of any degenerative changes.

In the *kidney*, which is also frequently affected, the change is similar to that in the liver. Here, also, it consists, for the most part, in an infiltration, with which may be associated the formation of roundish nodules and masses. Other organs are much less frequently involved—the lungs, pleuræ, stomach, intestines, and heart, may all, however, become the seats of the heteroplastic formations.

LYMPHADENOMA.—Closely allied to Leukæmia is the disease now generally known as *lymphadenoma*. This disease was formerly described by Hodgkin, Bright, Wilks, and Trousseau; and was called after the first-named of these observers, "Hodgkin's Disease." Trousseau designated it "Adénie." Recently its characteristics have been more fully determined by MM. Cornil and Ranvier, and by Drs. Murchison[1] and Sanderson, to whose descriptions the reader is referred for more complete information respecting it.

The disease consists in a hyperplasia of the lymphatic glands and of other lymphatic structures, together with a large formation of lymphatic tissue in various organs. Histologically, the new growths are precisely similar to those of leukæmia. The disease differs essentially, however, from leukæmia in this respect, that the new formation of lymphatic tissue is not associated with any increase in the number of the white corpuscles in the blood. The lymphatic glands are usually the earliest seats of the new growth. At first it may be limited to a single group of glands; the process, however, soon becomes more general, and the glands throughout the body become involved. They increase rapidly in size, so as to form large tumors, which are of a soft brain-like consistence. The Malpighian corpuscles of the spleen, as in leukæmia, also become enlarged and form grayish-white nodules and masses. Associated with these hyperplasias of the lymphatic structures, there is a large formation of lymphatic tissue in various organs: the liver, kidneys, lungs, stomach, muscle, bones, and subcutaneous tissue may all become the seats of the new growth. This either occurs as an infiltration, which in its distribution and physical characters is similar to that described as occurring in leukæmia; or as small nodules scattered through the substance of the organs. The heteroplastic growth appears to originate principally in the perivascular tissue: in the lungs, its chief seat is the tissue surrounding the minute bronchi. The migration of blood-corpuscles probably does not play such an important part in its production as it does in that of the leukæmic growths. The new tissue has but little tendency to undergo retrogressive changes, and it thus differs essentially from tubercle.

TUBERCLE.

The remaining variety of lymphoma—*tubercle*, will be considered in the following chapter.

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[1] "Trans. Path. Soc. Lond.," vols. xx and xxi.

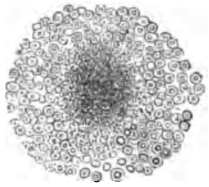
CHAPTER XX.

TUBERCLE.[1]

THE term "tubercle" was formerly applied indiscriminately to all pathological products which had undergone caseous degeneration, and which in their color and consistence somewhat resembled soft cheese. Caseation, however, as already stated, occurs not only in tubercle, but is a common result of the retrograde metamorphosis of many growths, which are destitute of or contain but few bloodvessels, and which consist of closely crowded cellular elements. (See "Caseation.") This change is indeed more characteristic of epithelial accumulations within the pulmonary lobules (catarrhal pneumonia), and of many growths in the lymphatic glands, and in osseous structures, than it is of tubercle itself.

The definition of "tubercle" has been rendered more precise by Virchow, who not only pointed out that all caseous masses were not tubercular, but established tubercle as a definite pathological growth, which, in its early stage, invariably consists of the grayish translucent nodule, known as the *gray granulation* or *miliary tubercle;* and to this the application of the term "tubercle" must be carefully restricted.

FIG. 30.



A Miliary Tubercle (diagrammatic). Showing the degeneration of the elements in the centre of the granulation.

The gray granulation or miliary tubercle is a grayish-white, translucent, non-vascular body of firm consistence, and well-defined spherical outline, and usually of about the size of a millet-seed. Although in its earliest stage it is uniformly translucent, its central portions quickly become opaque and yellowish, owing to the retrograde metamorphosis of its component elements. (Fig. 30.) These miliary tubercles are either isolated or grouped together into irregular masses. In the latter case, they are less distinct, and appear to be more or less confluent. Both the isolated tubercles and the larger masses are frequently surrounded by a zone of hyperæmia, or of pigment.

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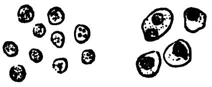
[1] It probably would have been more correct to have described tubercle amongst the *inflammatory* new formations. (See "Etiology.")

STRUCTURE.—In structure, tubercle, like the other lymphomata, consists of lymphatic cells contained in the meshes of a very delicate reticulum. The cells are most of them round, or roundly-oval, colorless, transparent and slightly granular bodies, much resembling lymph-corpuscles, and, like these, varying considerably in size: many of them contain a small, distinct nucleus. In addition to these there are a few larger cells, containing two or even three nuclei. (Fig. 31.) The nucleated cells are exceedingly destructible, so that often more free nuclei than cells are visible. The network within which these elements are inclosed, consists either of very delicate fibres, or of a more homogeneous transparent-looking tissue. In most cases it is so delicate, that it can only with difficulty be recognized, and the tubercle appears to consist almost entirely of closely crowded cells. Sometimes, however, it is more marked, and the granulation is then of a tough and more fibrous consistence.

FIG. 31.

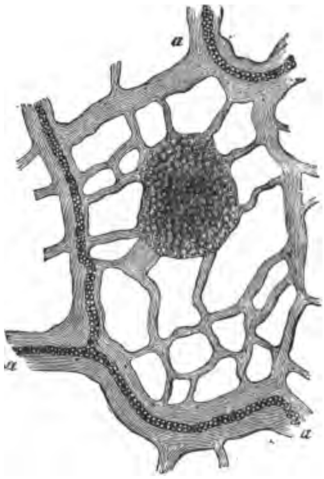


Cells from a Miliary Tubercle. × 400.

The granulation blends insensibly at its margins with the surrounding tissue. The cells in the external portions of the growth are more perfect than those in the centre, where, owing to the rapid retrograde metamorphosis which takes place, nothing is seen but a fine granular débris. (See Fig. 30.) Tubercle is essentially a non-vascular growth; in those masses, however, formed by the agglomeration of separate granulations, vessels are frequently met with. These do not belong to the tubercle, but are those of the part in which it is situated.

DEVELOPMENT.—Tubercle appears invariably to originate from tissues belonging to the lymphatic system. The tissue which surrounds the small arteries in many situations, constituting the lymphatic sheaths, is that from which it most frequently springs. The small cells in this situation multiply at separate centres, and thus are produced numerous miliary nodules around the vessel, which, as they develop, gradually compress, and may ultimately completely occlude it. This occlusion of the small vessels by the tubercular growth is very characteristic; it is well exemplified in the pia mater. (See "Tubercle in the Pia Mater.") Tubercle may, in the same way, originate from adenoid tissue in other situations—from that constituting the follicles of the spleen and lymphatic glands, from that which exists in the lungs in the neighborhood of the minute bronchi, from the small collections of it beneath the epithelium of the pleura and peritoneum, and from that beneath the mucous membrane of the alimentary canal. In other cases, the tubercle originates from cells situated within the lymphatic vessels. Many of the tubercular granulations which occur in the pleura and peritoneum, are simply overgrowths of minute nodules of adenoid tissue which normally exist in these situations. (Fig. 32.) When tubercle originates from the adenoid tissue surrounding the blood-vessels, and from that in the spleen and lymphatic glands, and in other situations where it exists in a diffuse form, the cell-proliferation takes place at several separate centres, so that numerous granulations are produced which may ultimately become more or less confluent at their margins, and so form larger masses. Such being the mode of development, it is evident that the tubercles are structurally inseparable from the tissue in which they grow, the centre of the granulation being surrounded by a zone of proliferating tissue.

FIG. 32.



A Miliary Tubercle of the Omentum. × 100. (Rindfleisch.)

Secondary Changes.—Tubercle invariably undergoes a retrogressive change; its elements being essentially unstable in their nature, are the seat of an early and rapid decay. This change commences in the centre of the granulations, and consists in the atrophy and incomplete fatty metamorphosis of the closely-crowded cellular elements, constituting what has been already described as *caseation*. The granulations—translucent and gray in their early stage—thus become opaque and yellowish, and the greater the amount of fat the more yellow is the color. Tubercle in this caseous condition was formerly regarded as a distinct variety, and was described under the name of "yellow tubercle." This, however, it must be borne in mind, is in all cases merely an advanced stage of the gray granulation. It is when this retrograde process has just commenced, and the tubercular nodule presents a grayish-white translucent margin, and a yellowish opaque centre, that it exhibits its most characteristic and distinctive features.

The caseous tubercle may subsequently soften, or gradually dry up into a firm cheesy mass, which may ultimately become calcified. Softening is much the most frequent result of the retrograde change. The caseous matter undergoes a process of liquefaction and becomes converted into a curd-like puriform liquid. If the growth is situated in a mucous membrane, the softened matters are eliminated and an ulcer is formed; if in the parenchyma of organs, as in the lungs or lymphatic glands, a pseudo-abscess results.

If the softened matters cannot be eliminated, the more fluid portions are absorbed and it gradually dries up into a firm caseous substance, which usually becomes infiltrated with calcareous particles and is thus converted into a hard cretaceous mass. In some cases the drying up and calcification take place without the caseous matter undergoing any antecedent process of liquefaction. The caseous and calcareous masses become encapsuled by fibrous tissue, and thus remain inert in the tissue, undergoing no further change.

In some cases this disintegration of the cellular elements is associated with an increase in the fibrillated reticulum of the growth. This gradually increases as the cells disappear, so that ultimately the tubercle may become converted into a mass consisting almost entirely of fibrous tissue, which contains in its meshes some of the débris of the original growth.

Etiology.—The views respecting the etiology and nature of tubercle have undergone great changes during recent years. Tubercle was formerly regarded, in accordance with the teaching of Laennec, as a specific non-inflammatory neoplasm, which like most other non-inflammatory growths, originated spontaneously in the tissues. More recently, however, observations have tended to show that in those cases in which *true* tubercle is most frequently met with—viz., in acute miliary tuberculosis, the growth is inflammatory in its nature, and owes its origin to the absorption and distribution of infective materials derived from some local lesion—in other words—that acute tuberculosis is of the nature of an *infective disease.* This view of the nature of tubercle was first promulgated by Buhl, who stated that in the majority of cases of this disease caseous masses existed in some part of the body, and that to the absorption of substances from these infective centres the general development of the tubercle was owing.

The results obtained from the experiments made to produce tubercle artificially in the lower animals, by Villemin, Wilson Fox, Burdon-Sanderson, Cohnheim, and others, tend greatly to support this view of its inflammatory nature. These results show that disseminated lesions, many of which are precisely similar to miliary tubercle as met with in man, may be produced either by the inoculation of various infective materials, or by the production of a local inflammatory induration; and that the disseminated growths result from the distribution of substances from the local source of infection by means of the bloodvessels and lymphatics. These cases of artificial tuberculosis, however, differ from the natural disease in this respect—that most of the disseminated lesions in the lungs and in other solid organs are not miliary tubercles, but consist of nodular and diffuse inflammatory growths, which, like the tubercle, quickly become caseous; whereas in the disease as it occurs in man, such inflammatory growths are usually almost entirely wanting. Notwithstanding this difference, there appears to be so close an analogy between the two conditions, that the infective nature of the one must be regarded as a strong argument in favor of the infective nature of the other.

Whilst therefore it is probable that acute miliary tuberculosis in most cases results from the distribution of infective materials from a local lesion, which is usually some pathological product which has become caseous—the question arises as to whether tubercle in a single organ or in a small portion of an organ, is owing to a similar cause. The tendency of evidence at the present day is to bring these localized tubercles also within the category of inflammatory growths. When occurring in the lungs, where they are most frequent, they rarely exist independently of caseous pneumonic products, and it would appear probable that their development here is usually secondary to the pneumonic process. (See "Pulmonary Phthisis.") In the lymphatic glands again, the development of tubercle is almost invariably a secondary process, resulting from the transmission of infective materials from some primary lesion—as tubercle of the mesenteric glands following tubercle of the intestine. In other situations, however, as in the intestines, larynx, and bronchi, the development of tubercle is not always thus to be explained; and although in these cases also, it is very frequently preceded and caused by some chronic irritation—as chronic intestinal or bronchial catarrh, the tubercle sometimes appears to be a *primary* and *non-inflammatory* growth.

Hereditary predisposition exercises a marked influence upon the development of tubercle. This predisposition, however, as stated by Niemeyer, probably consists more frequently in a tendency to chronic inflammations of certain organs, especially of the lungs, mucous membranes, and of lymphatic glands, than in a tendency to the development of *primary* tubercle; the localized or disseminated tubercular growths being secondary to and resulting from the direct irritation or infective properties of the inflammatory products.

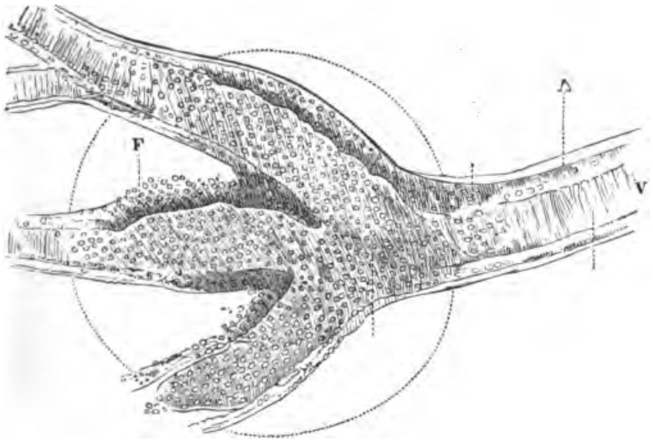
The organs in which tubercle is most frequently met with, arranged in the order of their frequency, are the lungs, the intestines, the lymphatic glands, the larynx, serous membranes, the pia mater, the spleen, the kidneys, and the liver. In many of these tubercle rarely occurs independently of general tuberculosis, viz.: in the pia mater, in the liver, in the spleen; in the kidneys, and in serous membranes.

TUBERCLE IN THE PIA MATER.

In the pia mater the growth of tubercle is associated with inflammation of the meninges, constituting the disease known as *tubercular meningitis.* This is almost invariably a part of general tuberculosis.

The tubercle is almost exclusively confined to the pia mater at the base of the brain, and is usually most abundant in the Sylvian fissures. A few scattered granulations are, however, frequently visible on the upper surface of the hemispheres. The growth originates in the perivascular sheaths which inclose the small arteries of the pia mater. (Fig. 33.) The cells within the sheath multiply, and the process of proliferation commencing at separate centres, numerous small gray granulations are produced around the vessel. These, which are distinctly visible to the naked eye, cause an external bulging of the sheath and a diminution in the calibre, or even complete obliteration of the inclosed vessel.

FIG. 33.



Miliary Tubercle in the Pia Mater. The dotted line indicates the original size of the tubercular nodule. A. The lymphatic sheath. V. The bloodvessel. F. Proliferation of elements within the sheath. × 100. (Cornil and Ranvier.)

The localized obstructions to the circulation which result from the pressure of the perivascular granulations, cause intense hyperæmia of the collateral vessels, and thus the pia mater at the base of the brain becomes exceedingly vascular; there being in some cases rupture of the vessels and extravasation. This is followed by an inflammatory process—a true basic meningitis. A transudation of liquor sanguinis takes place from the hyperæmic vessels, together with an escape of blood-corpuscles, and thus the meshes of the pia mater become infiltrated with a sero-fibrinous liquid, which in many parts has a puriform character.

These changes in the pia mater at the base of the brain are attended by softening of the immediately subjacent cerebral substance, which becomes infiltrated with young cells. The lateral ventricles at the same time become distended with serosity (acute hydrocephalus), so that the convolutions on the surface of the hemispheres are seen to be much flattened. The ependyma and choroid plexus also become exceedingly vascular, and the walls of the ventricles, together with the fornix and soft commissure, become much softened. All of these changes are owing, partly to an inflammatory process, and partly to the mechanical obstruction to the circulation caused by the tubercular growth. In addition, the arachnoid membrane is dry and sticky.

TUBERCLE IN THE LYMPHATIC GLANDS.

In the lymphatic glands, tubercle originates in the follicles of the gland; and as its development is owing to the presence of infective materials conveyed by the lymphatic vessels, the process usually commences in the follicles of the cortex. The process consists in the multiplication of the lymphatic elements, so that each tubercle is simply an enlarged lymph-follicle. The tubercles rapidly become caseous. They may increase so that the whole gland becomes involved, and is converted into a cheesy mass, which may soften, dry up, or calcify.

TUBERCLE IN MUCOUS MEMBRANES.

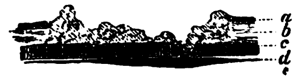
In mucous membranes the tubercular growth leads to ulceration. The intestinal, the bronchial, the laryngeal, and the uro-genital mucous membranes are those in which it is most frequently met with.

The Intestine.—In the intestine, the tubercular process is very similar to that in the lymphatic glands. It commences here in the solitary and Peyer's glands, and as in typhoid fever, it is especially these structures in the lower part of the small intestine and in the cæcum which are affected.

The first stage in the process consists in a hyperplasia and subsequent caseation of the lymphatic elements. In the solitary glands, the tubercle is simply an overcrowding of the elements of the gland, which quickly becomes caseous in its centre. In Peyer's patches, as in the lymphatic glands, the hyperplastic process commences in isolated follicles in the patch, so that the tubercles are seen as opaque yellowish nodules scattered over its surface. The tubercular process thus presents in its earliest stages a marked contrast to the typhoid one. (See "Typhoid Fever.")

The second stage in the process consists in the softening of the tubercle, and the formation of an *ulcer*. This, like the primary proliferation, commences in Peyer's patches at separate centres, and the ulcers extend at their margins, so that the whole patch may ultimately become involved. As the ulceration proceeds, the tubercular growth extends beyond the confines both of the follicles and patches, thus becoming in a certain sense, *heteroplastic*. This heteroplastic proliferation proceeds continuously at the margins and floor of the growth, whilst the ulceration is extending in the centre. It extends principally in the course of the bloodvessels, taking place in the perivascular tissue, and the growth may thus gradually involve the whole circumference of the intestine. It also extends deeply in the muscular coat, and may even reach the subserous tissue. The edges of the ulcer are thus thick and irregular, and the nodules of new growth are seen scattered in its floor. (Fig. 34.)

FIG. 34.



A Tubercular Ulcer of the Intestine (diagrammatic). *a.* Epithelial lining. *b.* Submucous tissue. *c.* Muscular coat. *d.* Peritoneum.

The process of ulceration takes place throughout the whole of the new growth, and it extends therefore beyond the confines of the follicles and patches, gradually implicating the whole circumference of the gut. The form of the ulcer thus contrasts strongly with that of typhoid, in which the ulceration rarely extends beyond the confines of the original patch. (See "Typhoid Fever.")

The tubercular ulcer rarely, if ever, heals. Its base becomes thickened by the new growth in the muscular and subserous tissues, and the thickening of the latter, together with that of the peritoneum itself, tends to prevent the occurrence of perforation. In the process of its extension the ulceration is attended by some contraction and narrowing of the gut.

TUBERCLE IN THE LUNGS.

Tubercle occurs in the lungs in *acute miliary tuberculosis*, and in many cases of *pulmonary phthisis*. In each of these diseases it must be considered separately.

Acute Miliary Tuberculosis.—This disease is characterized by a general development of tubercle not only in the lungs but in most other organs, and it appears, as already stated, to be in most cases of an infective nature, and to result from the transmission of infective materials from some caseous pathological product. It is in this disease that pulmonary tubercle is met with in its most characteristic form.

The tubercle originates here in two situations—around the bloodvessels, and in the neighborhood of the small bronchi. In the former, the growth consists in an accumultion of cells in the perivascular sheaths which inclose the small vessels. A proliferation of these cells takes place at separate centres, so that numerous miliary nodules are produced in the sheath: these may subsequently become more or less confluent. The effect of this perivascular growth is to compress the small bloodvessels, and the obstruction often extends for some distance beyond the confines of the nodular growths, owing to a hyperplastic process taking place in the cells of the vessels themselves.

In the neighborhood of the bronchi, the tubercle either originates in the minute masses of adenoid tissue which exist in the immediate vicinity of the bronchioles, or in the walls of the alveoli, in which no such structure exists. In whichever of these situations the growth takes place, it causes a diminution in the calibre of the terminal bronchi, and thickening of the alveolar wall.

This development of tubercle between the alveoli is soon followed by changes in the alveoli themselves. Not only do their walls become thickened, but they become the seat of a pneumonic process, which consists in a proliferation of their epithelium and its accumulation within the alveolar cavity (Catarrhal pneumonia). The effect of this is to cause nodules of pneumonic consolidation in various parts of the lung, which are usually intimately blended with the tubercular growths. These are larger than the tubercles, and less regular in outline, although in their earliest stages, they may present a similar translucent appearance. As they increase they become confluent, and so may form large tracts of pneumonic consolidation.

In most cases, however, of acute tuberculosis, this secondary pneumonic process does not take place, death ensuing before the tubercular growth has had time to cause changes in the alveolar epithelium. With the exception of any old caseous pneumonic product which may exist, the pulmonary tissue is usually but little altered, the lungs are crepitant throughout, being merely permeated with scattered miliary granulations, which may in some places be confluent in groups.

Pulmonary Phthisis.—The part which the development of tubercle plays in the production of pulmonary phthisis, is one of the most vexed questions in the pathology of the present day. In accordance with the teaching of Laennec, phthisis was formerly regarded in all cases as a *tuberculous* disease, and the consolidation and subsequent caseation and disintegration of the pulmonary tissue which are its essential anatomical features, were looked upon as invariably the result of the tubercular growth. The various consolidations of the pulmonary tissue were described as *infiltrated tubercle*, and tubercle in some form or other, was regarded as so essential a constituent of the disease, that "phthisis" and "pulmonary tuberculosis" came to be quite synonymous terms.

This old view of the tubercular nature of phthisis was principally based upon the erroneous notions which were then prevalent respecting the pathological significance of caseous products. Caseation was considered to be characteristic of tuberle, and all caseous masses were regarded as tubercular, hence phthisis—in which caseation is the most prominent feature—was regarded as a tuberculous disease. That such, however, is not the case, and that caseation is a form of metamorphosis more frequent in many other growths than in tubercle itself, has already been insisted upon.

When the application of the term "tubercle" became limited by Virchow and his followers to the gray granulation, it was evident that these old views respecting the nature of pulmonary phthisis, which dated from Laennec, were no longer tenable. In many cases of phthisis it was seen that no tubercle existed, and that in those cases in which it did exist, it was invariably associated with extensive consolidation and caseation of the pulmonary tissue. It soon became evident also, that although tubercle might be present in phthisical lungs, the principal part of the consolidation was owing, not to the tubercle but to a chronic inflammatory process. It then became a question in those cases in which tubercle did exist, as to the relation which subsisted between the tubercle and the pneumonic consolidation. The latter was, for the most part, regarded as secondary to the former, and the tubercle was still looked upon as the most important element in the disease.

During recent years, however, principally owing to the advocacy of Buhl, Niemeyer, and Waldenburg, there has been an increasing tendency to regard the tubercle met with in most phthisical lungs, as *secondary* to the pneumonic process; and to look upon the latter as playing the most important part in the production of pulmonary phthisis. According to these observers, the tubercle in most cases results from *infection* by the caseous pneumonic products, and phthisis is thus in the first place more frequently an *inflammatory* than a tuberculous disease.

Although it must now be admitted that chronic pneumonia, terminating in caseation, is the most frequent and important element in the production of pulmonary phthisis, and that tubercle, when it occurs, is usually a *secondary* growth,—it is probable that phthisis is by no means infrequently *primarily tubercular*, the pneumonic consolidation being *secondary* to the tubercular growth. As, however, all cases of phthisis are characterized by consolidation, which terminates in caseation and disintegration, the ultimate condition of the lung is much the same whether the disease be primarily pneumonic or tubercular in its nature.

The tubercle originates either in the perivascular sheaths, in the adenoid tissue in the neighborhood of the small bronchi, in the alveolar walls, or in the bronchial mucous membrane. When primary, it is most frequent in the last-named situation.

As the most important element in the production of pulmonary phthisis is chronic pneumonia, the further consideration of this disease will be postponed until the pneumonic process has been described. (See "Chronic Catarrhal Pneumonia.")

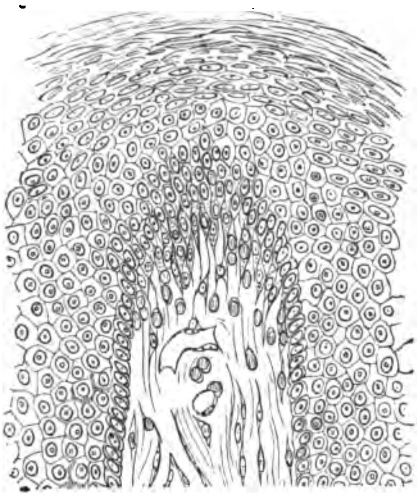
CHAPTER XXI.

THE PAPILLOMATA.

THE Papillomata are new formations resembling in structure ordinary papillæ, and like these they grow from cutaneous, mucous, or serous surfaces.

STRUCTURE.—They consist of a basis of connective tissue, supporting bloodvessels, which terminate in a capillary network or in a single capillary loop, the whole being enveloped in a covering of epithelium. (Fig. 35.) The epithelial covering varies in character in different growths. In those of the skin, it is like that met with in this situation, hard and stratified, and is usually very abundant, forming a dense firm covering. In those originating from mucous surfaces, the epithelium forms a thinner investment, and is of a much softer consistence; whilst in those growing from serous membranes it often constitutes only a single layer.

FIG. 35.



Papilloma. Showing a single enlarged papilla. (Rindfleisch.)

The growth may be simple—consisting merely of enlarged papillæ, as in a common wart—or it may be more complex, the papillæ being very numerous, and giving off secondary and tertiary offsets. If the investing epithelium is very abundant, it may so inclose the whole mass as to give to it a more or less regular outline. More commonly, however, this is not the case, and the epithelium not being sufficient to fill up the spaces between the papillæ, the growth presents a branched, villous, or cauliflower appearance. The bloodvessels are often very numerous, and are sometimes dilated and tortuous.

Development.—The papillomata always originate from the skin, from mucous, or from serous membranes. They most frequently grow from pre-existing papillæ; sometimes, however, they occur where no papillæ exist, springing directly from the subepithelial connective tissue;—this is the case in the stomach and larynx. Their growth is usually slow. The individual tumors rarely attain a very large size, the larger forms being for the most part constituted of several smaller growths.

Secondary Changes.—Of these, ulceration and hemorrhage are the most frequent. They occur especially in those growths which originate from mucous surfaces. The hemorrhage is often very abundant, and may even endanger life. This is not unfrequently the case in the papillary growths of the bladder and intestine.

Varieties.—The varieties of papillary tumors depend principally upon their seat. Those growing from the skin are *warts* and *horny growths*. These are firm, have a dense epithelial covering, and are less prone to ulceration and hemorrhage than those growing upon other parts. Larger and more vascular forms may, however, occur on cutaneous surfaces; such are the *condylomata* and *venereal warts* met with around the anus and upon the external male and female genital organs.

The papillomata of mucous membranes are softer and more vascular than the preceding, they have a less dense epithelial covering, and are more prone to ulceration and hemorrhage. Many of them come within the category of mucous polypi. They are met with on the tongue, in the larynx and nose, on the gastro-intestinal mucous membrane, on the cervix uteri, and in the bladder. In the bladder and intestine they are often exceedingly vascular, and give rise to profuse hemorrhage.

Here they are not unfrequently confounded with villous epithelial cancer.

Papillomata of serous membranes never form distinct tumors. They are met with as small outgrowths from the synovial membrane in chronic diseases of joints.

Clinical Characters.—Clinically, the papillomata are innocent growths. They may, however, prove fatal from continuous ulceration and hemorrhage: this is especially the case, as already mentioned, in papilloma of the bladder and intestine. In these situations they are easily mistaken for villous cancer; the symptoms of both are very similar, and it is often only after death that they can be distinguished. In the papillomata the epithelium is *homologous*, being situated only *upon the surface* of the papillæ, and in no case growing *within* their connective tissue basis. In the cancers, on the other hand, it is *heterologous*, and is met with in the basis of the tumor and in the subjacent connective tissue. (See "Epithelioma.")

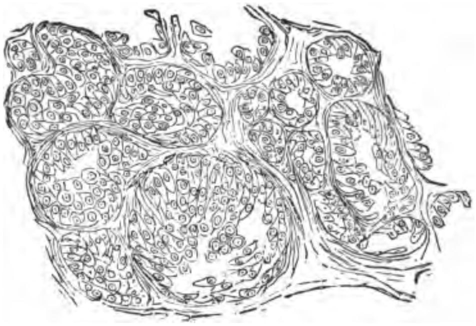
CHAPTER XXII.

THE ADENOMATA.

THE Adenomata, or, as they are more commonly called, *glandular tumors*, are new formations of gland-tissue.

STRUCTURE.—In structure the adenomata resemble the racemose or tubular glands. They consist of numerous small saccules or tubes filled with squamous or cylindrical epithelial cells. These are grouped together, being merely separated by a small, though varying, amount of connective tissue, in which are contained the bloodvessels. (Fig. 36.)

FIG. 36.



Adenoma of the Mamma. x 300. (Rindfleisch.)

DEVELOPMENT.—The adenomata always originate from preexisting gland-structures, of which they are simply local hyperplasias. Their growth, which is usually slow, takes place by the development of diverticula from the saccules or tubules of the gland, and by a proliferation of the inclosed epithelium. The new growth may remain in intimate relation with the adjacent gland, or it may ultimately become separated from it by a fibrous capsule.

SECONDARY CHANGES.—The most frequent of these is fatty degeneration of the epithelium, which may give rise to the formation of small caseous masses in the growth. Dilatation of the saccules and tubules so as to form cysts, and mucoid softening, are also common.

Varieties.—One of the seats of adenoma is the mammary gland. Here two varieties must be distinguished—one, in which there is a general hyperplasia of the whole gland; the other, in which the process is limited to a single, or to a small group of lobules. The former constitutes hypertrophy of the mamma, the latter is the *chronic mammary* or *adenoid* tumor. Adenoma of the mamma is comparatively rare. Many growths in this situation, described as adenomata, are sarcomatous tumors, in which are contained the acini of the gland. The distinction between true adenoma and such sarcomatous growths is often exceedingly difficult. The adenomata are either superficial or deeply seated in the gland from which they originate, and from which they are usually separated by a loose fibrous capsule. They are commonly round or oval in shape, lobular, and of a hard elastic consistence. On section they present a lobulated appearance, their racemose structure being often visible to the naked eye.

The glandular structures of mucous membrane are also very common seats of adenoid growths. In the nose, the pharynx, the stomach, the intestines, the vagina and uterus, these growths are frequently met with. In course of time they usually gradually project above the surface of the membrane, so as to form a polypus, and thus constitute the most common form of *mucous polypi*. In consistence they are soft and somewhat gelatinous, and often present a semi-translucent appearance. Their surface resembles in color the surrounding mucous membrane. The formation in them of cysts, by the dilatation of their tubules, is exceedingly frequent; the cysts usually contain a soft mucoid substance.

The other seats of adenomata are the sebaceous glands, the pineal gland, and the ovaries.

Clinical Characters.—Clinically, the adenomata are for the most part perfectly innocent; they are, however, very liable to be confounded with growths possessing malignant properties. A tumor, also, which is primarily a simple adenoma, may subsequently become cancerous. The anatomical distinction between cancer of a gland in its earliest stages, and a simple glandular tumor is often exceedingly difficult, especially in the mamma. In cancer the growth commences by a proliferation of the epithelium within the ducts of the gland; and as the epithelium only subsequently becomes heteroplastic, the determination of the nature of the tumor in this stage, is necessarily attended with considerable difficulty. In sarcomatous tumors, again, originating in the connective tissue of a gland, the ducts of the gland filled with epithelium, are often seen imbedded in the new growth, and thus the appearance of adenoma may be closely simulated.

CHAPTER XXIII.

THE CARCINOMATA.

THE Carcinomata, or Cancers, are new formations consisting of cells of an epithelial type, without any intercellular substance, grouped together irregularly within the alveoli of a fibrous stroma.

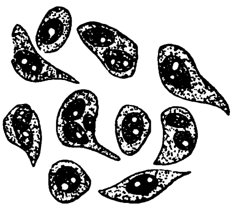
The term "*cancer*" has been so commonly applied indefinitely to any growth possessing malignant properties, that "*cancerous*" and "*malignant*" have come to be regarded by many as synonymous terms. It is important, however, clearly to distinguish between them. A *cancer* is a growth possessing the above-named definite structure; a *malignant* growth, on the other hand, is one which, independently of its structure, is infectious. (See "Malignancy.") "Cancerous," is an *anatomical* term; "Malignant," is a *clinical* one.

The Cancers include the four following varieties: *Scirrhus*, *Encephaloid*, *Epithelioma*, and *Colloid*. These, although all possessing the same general characters, present certain structural and clinical differences which serve to distinguish them. It will be well, in the first place, to describe the characters common to the whole class, and subsequently those which are peculiar to the individual members.

STRUCTURE.—In structure, the Cancers so far resemble one another, that they all consist of cells of an epithelial type, without any intercellular substance, grouped together irregularly within the alveoli of a fibrous stroma.

The *cells* are characterized by their large size, by the diversity of their forms, and by the magnitude and prominence of their nuclei and nucleoli. (Fig. 37.) In size they vary from $\frac{1}{300}$ to $\frac{1}{1500}$ of an inch in diameter; the majority being about five times as large as a red blood-corpuscle. They are round, oval, fusiform, caudate, polygonal—exhibiting, in short, every diversity of outline. These variations in form are principally owing to

FIG. 37.

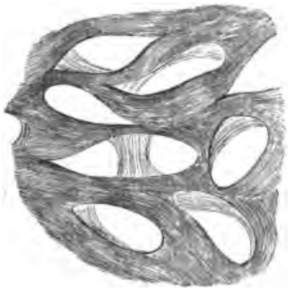


Cells from a Scirrhus of the Mamma. × 250.

the mutual pressure to which in their growth they are subjected. The nuclei, which are large and prominent, are round or oval in shape, and contain one or more bright nucleoli. The nuclei are, perhaps, most frequently single; two, however, are frequently met with, and in the softer and more rapidly growing cancers they may be much more numerous. The cells rapidly undergo retrogressive changes, hence they usually contain molecular fat. They are, many of them, exceedingly destructible, so that often more free nuclei than cells are visible. Cells precisely similar to these are met with in other morbid growths, and even in the normal tissues. There is thus no *specific* " cancer-cell." It is the general character of the cells, together with their mode of distribution in the meshes of a fibrous stroma, that determines the nature of the growth to which they belong.

The *stroma* varies considerably in amount, being much more abundant in some varieties of cancer than in others. It consists of a fibrillated tissue arranged so as to form alveoli of various forms and sizes, within which the cells are grouped (Fig. 38). These alveoli communicate with one another so as to form a continuous cavernous system. The characters of the stroma vary with the rapidity of its growth; if this is rapid, it will contain numerous round and spindle-shaped cells; if, on the other hand, it is slow or has altogether ceased, the tissue will contain but few cells, and will be dense and fibrous in character. This is the condition in which it is most commonly met with.

FIG. 38.



Stroma from a Scirrhus of the Mamma. × 200.

Within the stroma are contained the *bloodvessels.* These are often very numerous, and form a close network. They are always limited to the stroma, and in no case do they encroach upon the alveoli. This distribution of the bloodvessels is important as distinguishing the cancers from the sarcomata; in the latter the vessels are not supported by a stroma, but ramify amongst the cells of the growth, hence the facility with which these tumors become generally disseminated.

In addition to the bloodvessels, the cancers also possess *lymphatics*. These accompany the bloodvessels, and, as has been shown by MM. Cornil and Ranvier, communicate with the alveoli; hence the great tendency of cancer to infect the lymphatic glands.

DEVELOPMENT.—The question of the genesis of cancer, involves that of the genesis of epithelium generally. It is maintained by many histologists that epithelium can only originate from epithelium, and that the strata of cells set aside in the embryo for the production of the epithelial tissues, is the source from which all epithelium is subsequently derived. Others admit that epithelium may also originate from connective tissue. A like difference of opinion exists as to the source of the epithelial cells in cancer. By many—as Waldeyer, Thiersch, and Billroth—they are regarded as originating only from pre-existing epithelium. Others—amongst whom are Virchow, Lücke, Rindfleisch, and Klebs—maintain that they may also be derived from the cells of connective tissue (or from migrated white blood-corpuscles).

The difficulty of determining the genesis of cancer, is partly owing to the fact that it so frequently originates in structures where epithelium is normally abundant, as in the mamma, skin, and alimentary canal; and that this normal epithelium is always, from the earliest stage of the growth, the seat of active proliferation. In cancer of the mamma, for example, the first change usually observable is a large accumulation of epithelium within the ducts of the gland, a condition similar to that already described as occurring in the development of an ordinary glandular tumor. (See "Adenoma.") Soon, however, the epithelium is found outside the ducts, amongst the inter- and periglandular connective tissue, which is also in a state of active proliferation, and is infiltrated with small round cells. The outline of the ducts ultimately becomes completely annihilated, and the epithelial cells are seen in alveoli, formed of a fibroid tissue. The question arises as to whether the epithelial cells, which constitute the ultimate cancerous growth, originated from those normally existing within the ducts of the gland, which in the process of development have extended beyond the ducts into the surrounding tissue; or whether they originated in the connective tissue—the proliferation of the glandular epithelium being merely a secondary process, and resulting from the irritation of the interglandular growth.

In those cases in which cancer occurs primarily in situations where no epithelium normally exists, as in the medulla of bone, and in lymphatic glands, it is difficult to maintain an epithelial origin. It must be admitted, however, that the tendency which is exhibited by the cells of cancerous growths to maintain the type of the epithelial structures in the vicinity of which they grow, is greatly in favor of the view that they are derived from the epithelium. In cancers situated near the cutaneous surfaces, for example, the cells are usually of the squamous type, whereas in those growing in connection with the glands of mucous membranes, they more commonly resemble the epithelium of the gland. It must be borne in mind, also, that the characters of young epithelium are by no means well defined. It is impossible to draw a line of demarcation between young epithelial cells, and the small round cells which are so numerous in the growing stroma of the cancer. It would appear to be quite in accordance with our present state of knowledge to admit the probability of two different modes of origin of the epithelial cells in cancer—one from epithelium, and the other from connective tissue; in the former case the cells being more strictly epithelial, such as are met with in epithelioma; in the latter, the epithelial characters being less marked, as in many forms of encephaloid and scirrhus.

The *stroma* is partly a new growth, and partly the pre-existing connective tissue of the part in which the cancer originates. It is probably in the main a new formation, and the pathology of its development will vary with that of the origin of the epithelial elements of the cancer. If the latter originate from epithelium, the stroma may be regarded as the result of the irritation of the connective tissue by the infiltrating epithelial cells. If, on the other hand, the cellular elements originate in connective tissue, the stroma must be regarded as a portion of the product of the connective tissue hyperplasia.

The cancers in their growth very rarely become encapsuled, but gradually invade the surrounding structures. This process of invasion is very characteristic, and is more marked in cancer than in any of the malignant growths. The epithelial elements are seen infiltrating the tissues for some distance around the confines of the tumor, so that there is no line of demarcation between it and the normal structures.

SECONDARY CHANGES.—The most important of these is fatty degeneration. This always occurs to a greater or less extent in all the varieties of cancer. The more rapid the growth, the earlier does this retrogressive change take place, and the greater is its extent; hence it is usually most marked in encephaloid. It produces softening of the growth, which is often reduced to a pulpy cream-like consistence. Caseation, pigmentation, mucoid and colloid degeneration may also occur. Calcification is very rarely met with.

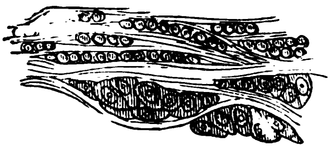
VARIETIES.—The term "cancer" was so vaguely applied by the older pathologists—nearly all malignant formations being included under this head—that considerable confusion has resulted in the classification of cancerous growths. Peculiarities in structure and appearance have given rise to special names, hence the terms—"*osteoid*," "*chondroid*," "*cystic*," "*villous*," and "*fungoid*" cancer. A cancer containing large quantities of pigment was described as a distinct variety, under the name of *melanotic* cancer. This pigmentation is most common in encephaloid.

The most convenient classification, and that which is now generally adopted, divides the cancers into four groups: *scirrhous*, *fibrous*, or *chronic* cancer; *encephaloid*, *medullary*, or *acute* cancer; *epithelioma*, or *cancroid;* and *colloid* or *gelatiniform* cancer. This division is based principally upon the relative proportion of the stroma, and upon the type of the epithelial elements.

SCIRRHOUS CANCER.

Scirrhous, *fibrous*, or *chronic* cancer is characterized by the large amount of its stroma and by the chronicity of its growth. The slowness in the development of scirrhus probably accounts in great measure for the peculiarities in its structure and physical characters.

FIG. 39.

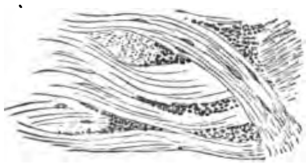


Scirrhus of the Mamma. Showing the development of the Epithelial Cells within the alveoli of the Stroma. × 300. (Rindfleisch.)

The epithelial growth, although at first it may be luxuriant, quickly subsides. The elements soon atrophy and undergo retrogressive changes. They are most abundant in the external portions of the tumor, where growth is taking place; in the central portions they may be almost entirely wanting. (Fig. 39.)

This degeneration of the epithelial elements is probably owing to the excessive growth of the stroma, and to the subsequent induration and contraction which it undergoes. It quickly assumes the characters of cicatricial tissue, and becomes hard and indurated. This causes obstruction and obliteration of the bloodvessels which it contains, and it is probably to this interference with the vascular supply that the arrest in the development of the cancer is owing. The whole of the central portions of the growth may thus ultimately consist simply of dense fibrous tissue, the circumference being the only part where the epithelial structure is visible. (Fig. 40.) The amount of atrophy and contraction varies considerably in different cases.

FIG. 40.



Scirrhus of the Mamma. Showing the cicatricial tissue in the older portions of the growth. × 300. (Rindfleisch.)

The physical characters of scirrhus are in the same way due to the abundance of its stroma. The growth is firm and hard, and it is often depressed in the centre, owing to the contraction of the cicatricial tissue: this is very characteristic of scirrhus of the breast, where it causes puckering of the superjacent structures. On section the tumor presents a white glistening surface, intersected with fibrous bands. The external are less firm than the central portions of the growth, and yield on scraping a juice which is rich in cells, nuclei, and granules.

Scirrhus is most commonly met with in the female breast, and in the alimentary canal—especially in the pylorus, œsophagus, and rectum. It also occurs in the skin.

ENCEPHALOID CANCER.

Encephaloid, *medullary*, or *acute* cancer, is closely allied to the preceding, from which it differs principally in the great rapidity of its growth, the small amount of its stroma, and the consequent softness of its consistence. Encephaloid and scirrhus cannot be regarded as constituting distinct varieties of cancer. There are all intermediate stages between them, and the differences in the rapidity of their growth, and consequently in their structure and physical characters, constitute their only distinctive features.

The epithelial growth in encephaloid is rapid and abundant, and the cells quickly undergo fatty degeneration, so that often more free nuclei than cells are visible. The proportion of stroma is very small, and owing to the rapidity of its growth, it is much less fibrous than that of scirrhus, and does not undergo a similar cicatricial contraction. The bloodvessels are very abundant, and the tissue supporting them being soft and nonresistant, hemorrhage readily takes place.

Encephaloid cancer is of a soft brain-like consistence, the central portions, where fatty degeneration is most advanced, often being completely diffluent. The tumor is often more or less lobulated. On section, it presents a white pulpy mass, much resembling brain-substance, which is often irregularly stained with extravasated blood.

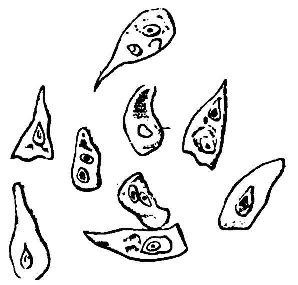
Encephaloid is most frequently met with in internal organs, as a *secondary* growth. It also occurs primarily, in the articular ends of bones, in the eye, in the testis, and in other parts. Many growths formerly described as encephaloid cancer, are soft Sarcomata. (See " Round-celled Sarcoma.")

EPITHELIOMA.

Epithelioma, *cancroid*, or *epithelial cancer*, must be regarded as constituting a much more distinct variety of cancer than either of the preceding. It differs from these in always growing in connection with cutaneous or mucous surfaces, and in its epithelial elements closely resembling the squamous variety of epithelium.

The cells of epithelioma are in the main indistinguishable from those met with on the cutaneous surfaces, and on the mucous membrane of the mouth. They vary in size from $\frac{1}{300}$ to $\frac{1}{1000}$ of an inch in diameter, the average being $\frac{1}{700}$. They contain usually a single nucleus; frequently, however, the nuclei are multiple. (Fig. 41.) They are often considerably flattened and distorted in shape, owing to the pressure to which in their growth they are subjected. The arrangement of these cells is peculiar: the majority of them are situated in irregular tubular-shaped lobules; others are less regularly grouped in masses of various sizes amongst the meshes of the stroma. As the cells increase in number they become arranged concentrically in groups, so as to form globular masses. In these masses, as the epithelium multiplies, the peripheral layers of cells become flattened by pressure against the surrounding structures, whilst those in the centre remain more or less spherical in shape, like those of the deeper layers of the epidermis. These are the *concentric globes*, or *epithelial nests*, which are so characteristic of epithelioma. (Fig. 42.) They are met with not only in the tubular lobules, but also in other parts of the stroma. The cells may be so closely packed as ultimately to become hard and dry like those of the nails and hair, and the globes are then of a brownish-yellow color, and of a firm consistence. These globes are often large enough to be readily visible to the naked eye, and owing to the arrangement of the epidermic scales, they usually present a fibrous appearance.

FIG. 41.



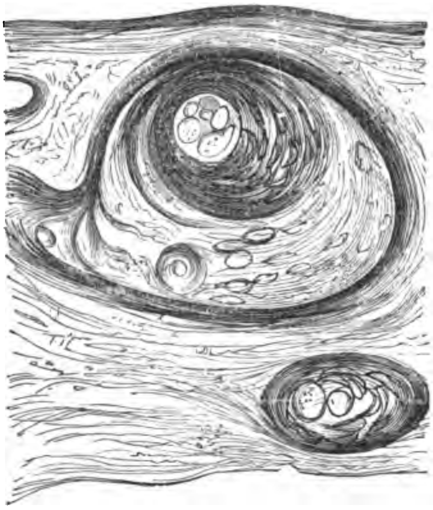
Cells from an Epithelioma of the Lip. x 250.

The stroma presents every variation between rapidly growing embryonic and dense fibrous tissue. It may be very abundant, or almost entirely wanting. As in the other forms of cancer, it supports the bloodvessels.

With regard to the development of epithelioma, there can be no doubt that its epithelial elements are derived from the epithelium of the skin or mucous membranes, or from that of the glands which are situated in these tissues. The growth commences by a proliferation of this epithelium, which as it increases becomes *heterologous*, extending beyond the normal limits into the subjacent connective tissue, and even into muscle, bone, and other structures.

Epithelioma usually presents itself, in the first place, either as a small foul ulcer with indurated edges, or as a subcutaneous induration or nodule, which subsequently ulcerates. The surface of the ulcer is frequently papillated and villous, owing to the irregular growth of the corium. The tumor itself is firm in consistence, often more or less friable, and on section presents a grayish-white granular surface, intersected with lines of fibrous tissue. The cut surface yields on pressure a small quantity of turbid fluid, and in most cases also a peculiar, thick, crumbling, curdy material can be expressed, which comes out in a worm-like shape, like the sebaceous matter from the glands of the skin. This latter is very characteristic; it is composed of epithelial scales, and on being mixed with water it does not diffuse itself like the juice of other cancers, but separates into minute visible particles. If it is very abundant, the cancer is soft and friable, and the material can be seen in the cut surface as small, scattered opaque dots.

FIG. 42.



Epithelioma of the Lip. Showing the concentric globes of epithelial cells. × 100.

Epithelioma has its primary seat in the immediate vicinity of the cutaneous or mucous surfaces; and it appears in many cases to owe its origin to some external source of irritation. It is most frequently met with in the lower lip at the junction of the skin and mucous membrane, on the tongue, prepuce, scrotum ("chimney-sweeps' cancer") labia, eyelids, cheeks, and in the uterus and bladder. As it extends it may involve any tissue—lymphatic glands, muscle, bone, and tendon may be alike implicated. It very rarely occurs in internal organs.

Epithelial cancers growing from mucous membranes which possess a cylindrical epithelium, differ somewhat from the preceding. They usually originate in the glandular structures of the membrane, and their epithelial elements resemble those of the gland, and are not squamous in character. There is rarely a formation of concentric globes, and the growths are of a soft, and often gelatinous consistence. The distinction between these and simple adenomata is often exceedingly difficult.

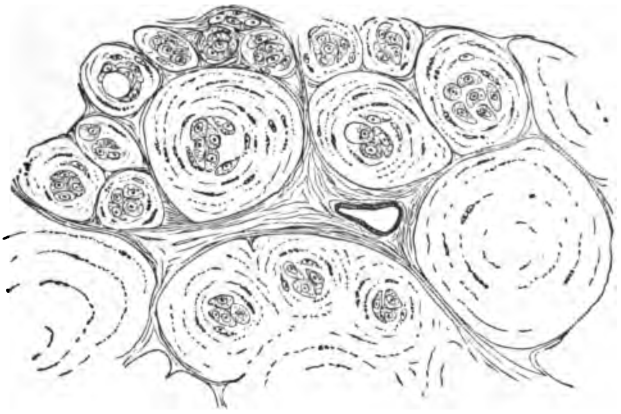
COLLOID CANCER.

The growths described under the name of *colloid*, *alveolar*, or *gelatiniform* cancer, although often regarded as constituting a distinct variety of cancer, are probably simply one of the preceding forms which have undergone a mucoid or colloid change. The frequency with which non-cancerous growths which have undergone these forms of degeneration have been confounded with colloid cancer has already been alluded to. (See " Colloid Degeneration.")

The alveolar structure in colloid cancers is very marked: the alveoli are large, distinct, and more or less spherical in shape. Within them is contained the gelatinous or colloid material, which is a glistening, translucent, colorless, or yellowish substance, of the consistence of thin mucilage or size-gelatin. In the main, it is perfectly structureless; within it, however, are imbedded a varying number of epithelioid cells, which also contain the same gelatinous substance. These cells present a peculiar appearance: they are large and spherical in shape, and are distended with drops of the same gelatinous material as that in which they are imbedded. (Fig. 43.) Many of them display a lamellar surface, their boundary consisting of concentric lines. It would appear that the mucoid change commences in the cells, which become gradually destroyed in the process.

Colloid cancer is most frequently met with in the stomach, in the intestine, and in the peritoneum.

FIG. 43.



Colloid Cancer. x 300. (Rindfleisch.)

CLINICAL CHARACTERS OF THE CANCERS.—The cancers all possess, in the highest degree, malignant properties. They extend locally, invading indiscriminately the tissues in which they grow, and reproduce themselves in the lymphatic glands and in internal organs. In the process of dissemination, however, they present some peculiarities which distinguish them from growths, with which they are closely allied, and which are equally malignant—viz., the sarcomata.

The cancers are characterized by their great tendency to local extension by the invasion of adjacent structures, and by the facility with which they cause similar growths in the neighboring lymphatic glands. The implication of the lymphatics is much more marked than in the sarcomata, and this is probably owing to the communication of these vessels with the alveolar spaces of the cancerous growth. The general dissemination in internal organs, on the other hand, is effected much less readily in cancer than in sarcoma, and the course of the former is therefore often more protracted than that of the latter. This difference is explained by the difference in the distribution of their bloodvessels; in cancer, these are contained in the stroma, and do not come into contact with the cells of the growth, whereas, in the sarcomata, they ramify amongst the cells, and their walls being composed of thin, embryonic tissue, like that of the growth which they supply, infection through the medium of the blood is rapidly and readily effected. In cancer, the lymph being the chief medium of infection, the reproduction of the growths in internal organs is often considerably delayed. The progress of the disease becomes arrested by the lymphatic glands, and its further dissemination can usually only be effected after these have become very generally and extensively involved.

With regard to the difference in the clinical characters of the several varieties of cancer—epithelioma is the least malignant. It extends locally, and affects the neighboring lymphatics, but rarely reproduces itself in internal organs. This is possibly partly owing to the size and character of its epithelial elements.

Scirrhus and encephaloid, which anatomically are so closely allied, differ considerably in the degree of their malignancy. The dissemination of the latter takes place much more readily than that of the former, owing to the greater rapidity of its growth, its greater vascularity, and the greater activity of its epithelial elements. Both scirrhus and encephaloid reproduce themselves in the lymphatic glands, and in internal organs.

Colloid closely resembles encephaloid in the rapidity of its development, and in the degree of its malignancy.

In all the varieties of cancer there is a tendency for the secondary growth to repeat the characters of the primary one. This is most marked in encephaloid, epithelioma, and colloid. These, in reproducing themselves, almost invariably maintain their primary characters. In scirrhus, however, the secondary growths in internal organs usually differ from the primary one. They are soft and vascular, and possess all the characters of encephaloid. A tendency to the formation of pigment (melanosis) in the primary cancer, is in the same way exhibited by the secondary growths. In some cases, however, this tendency to repeat the characters of the primary growth is less marked, and the different varieties of cancer may be found replacing one another.

CHAPTER XXIV.

THE MYOMATA, NEUROMATA, AND ANGIOMATA.

MYOMATA.

THE Myomata are tumors consisting of muscular tissue. A new formation of muscle has been already described as being frequently associated with the ordinary process of hypertrophy, both of striated and of non-striated muscle—a simple hyperplasia of the elements of the muscle accompanying the increase in their size. (See "Hypertrophy.")

STRUCTURE.—The myomata consists either of striated or of non-striated muscle. The former are exceedingly rare, only two or three examples having been recorded, and these were congenital.

The myomata of *non-striated* muscle, consist—like the physiological tissue—of elongated, spindle-shaped cells, more or less isolated, or grouped into fasciculi of various sizes, with a varying quantity of connective tissue. The connective tissue is often exceedingly abundant, so much so, that these growths in the uterus—where they most frequently occur—are usually known as *fibroid tumors.* It is often necessary to macerate the tissue in dilute nitric acid, in order to display the muscular elements, which by this means become isolated. The muscular fasciculi either present a regular arrangement, or pass in different directions through the tumor. The bloodvessels are distributed in the connective tissue.

DEVELOPMENT.—The myomata probably always originate from muscle, they are therefore homologous growths. In their growth they may become distinctly circumscribed tumors, but more commonly, they remain as ill-defined, irregular masses in the midst of the muscular tissue in which they grow. They not infrequently become pedunculated and form polypi, especially in the uterus.

SECONDARY CHANGES.—Of these, the most frequent is calcification; hemorrhage, mucoid softening, and the formation of cysts, are also occasionally met with.

The *uterus* is by far the most frequent seat of the myomata, and they constitute here a variety of the so-called *fibroid* tumors of the uterus. Many of these uterine fibroid tumors consist almost entirely of connective tissue, and to such the application of the term "*fibroid*" would appear to be correct. In others, however, the muscular elements form so large a proportion of the tumor that it is more properly regarded as a muscular growth. They form either distinctly circumscribed tumors, or irregular, ill-defined masses in the uterine walls. Sometimes they project in the form of polypi into the uterine or abdominal cavities. Myomata may also occur in the prostate gland, in the œsophagus, and in the stomach and intestines. In the latter situations they often become pedunculated.

CLINICAL CHARACTERS.—Clinically, the myomata are perfectly innocent.

NEUROMATA.

The neuromata are tumors consisting almost entirely of nerve-tissue. The term "neuroma" has been applied to many growths found in connection with nerves: fibrous and gummy tumors growing within the nerve-sheath, and many myomata, have been included under this head. True neuroma, however, is very rarely met with, and is amongst the least frequent of all the new formations.

STRUCTURE.—Nerve-tissue presents itself in two forms: the gray or medullary tissue, which consists principally of nerve-cells, and the white tissue, which consists of the tubular nerve-fibres. New formations of medullary tissue have been described by Virchow in the nervous centres, as local or general hyperplasias of the gray matter, but they are so exceedingly rare that a further description of them will be unnecessary. It is as a new growth of *nerve-fibres* that the neuromata are most frequently met with. They resemble in structure the cerebro-spinal nerves, consisting of tubular fibres with a varying quantity of intertubular connective tissue, and in some cases a few gray gelatinous fibres.

DEVELOPMENT.—The neuromata always originate from pre-existing nerve-tissue,—either from the cranial or from the spinal nerves. Their growth is slow, they rarely attain a large size, but usually exist as small, single nodules.

The most frequent seat of these growths is the extremities of divided nerves, where they are sometimes found after amputations. They exist, in this situation, as spherical or elongated enlargements of the divided extremity of the nerve; and they are usually intimately connected with the cicatricial tissue of the stump, from which they can only with difficulty be isolated. They may also occur in the course of the nerves in any situation, either as single or multiple nodules.

CLINICAL CHARACTERS.—Clinically, the neuromata are for the most part perfectly innocent tumors. They sometimes, however, recur locally after removal. They always cause considerable pain.

ANGIOMATA.

The angiomata, or vascular tumors, are tumors consisting of bloodvessels, held together by a small amount of connective tissue. They include the various forms of nævi, the erectile tumors, and aneurism by anastomosis. They may be divided into two classes, the *simple* angiomata, in which the new vessels resemble normal arteries, veins, or capillaries; and the *cavernous* angiomata, in which the blood circulates in a cavernous structure similar to that of the corpus cavernosum penis.

SIMPLE ANGIOMATA.—These include the various forms of nævi, and telangiectasis. They consist of tortuous and dilated bloodvessels, held together by a small quantity of connective and adipose tissue. The vessels are most of them of new formation; some, however, may be the original vessels of the part which have become considerably enlarged. They most commonly partake of the nature of capillaries, but in other cases the arterial or venous characters predominate. These growths are usually small, superficial, slightly elevated masses; although they sometimes form larger tumors. Their color is red, violet, or purple, according to the character of the blood which they contain. The former is much the most frequent.

CAVERNOUS ANGIOMATA.—These include the venous vascular tumors, erectile tumors, and aneurism by anastomosis. They consist of an erectile cavernous tissue, closely resembling that of the corpus cavernosum penis. The growth is made up of irregular fibrous alveoli, which communicate freely with one another, and are lined with an epithelium similar to that of the veins. These spaces are distended with blood, usually venous, which is supplied to them by numerous tortuous vessels and circulates in them with varying degrees of rapidity. These growths are commonly of a bluish color. They may be diffuse, or form distinctly circumscribed tumors. They often exhibit distinct pulsation. Their favorite seat is the skin and subcutaneous tissue. They may also occur in the orbit, in muscle, and in the liver, spleen, and kidneys.

CHAPTER XXV.

CYSTS.

In addition to the new growths already described, there is a large class of formations, many of which cannot be regarded as "tumors" in the strict application of this term. These are the *cysts* or *cystic tumors.*

A *cyst* is a cavity containing liquid or pultaceous material, which is separated from the surrounding structures by a more or less distinct capsule. It may be a new formation, or a pre-existing structure which has become distended by its own secretion, or by extravasation into it. The former only comes within the category of new growths, although, for the sake of convenience, it will be advisable to consider them both under one head.

There are thus two principal modes by which cysts originate; one, the most frequent, by the gradual accumulation of substances within the cavities of pre-existing structures, which are, for the most part, products of their own formation, being in some cases a secretion, and in others a cell-growth; the other, by the independent formation of a cyst in the tissues.

The accumulation of secretions and of other products within pre-existing cavities, may be effected in the three following ways:

1st. By the retention of the normal secretion, owing to the closure of the excretory ducts, as in sebaceous glands.

2d. By excessive secretion; the cavity being unprovided with an excretory duct, as in bursæ.

3d. By the extravasation of blood into the cavity, as in hæmatocele.

The independent formation of a cyst may take place—

1st. By the softening and liquefaction of the tissues in some particular part, owing to mucoid or fatty changes. The tissues around the softened matters become condensed, and ultimately form a kind of cyst-wall.

2d. By the enlargement and fusion of the spaces in connective tissue, and the accumulation of fluids within them. The surrounding tissue becomes condensed and forms a cyst-wall; and this may, in some cases, become lined with secreting cells.

3d. By the formation of a cyst-wall around foreign bodies, parasites, or extravasated blood.

STRUCTURE.—The wall of the cyst will vary in its nature according as it is a pre-existing or a newly-formed tissue. In the former case it will possess an epithelial lining, which will present the same characters as that of the gland, serous membrane, or other structure, from which the cyst originated. If the cyst is a new growth it rarely possesses an epithelial lining, but consists simply of a fibrous capsule. The cyst-wall is sometimes firmly connected with the adjacent parts, so that it can only with difficulty be separated; in other cases the union is much less intimate. Instead of being a distinct structure, it may simply be the surrounding tissue which has become dense and fibrous in character.

The contents of cysts are very various, and may serve as a basis for their classification. In the retention-cysts they will vary with the nature of the normal secretion—serum, sebaceous matter, saliva, milk, seminal fluid, and other substances are thus found in these cysts, more or less altered in character from being retained in a closed cavity. In the exudation-cysts serum is the most frequent constituent, and in extravasation-cysts, blood. In those cysts which originate from the softening and breaking down of tissue, the contents are the products of retrogressive tissue metamorphosis, and usually consist largely of mucin, fatty matters, and serum.

SECONDARY CHANGES.—These may take place in the wall of the cyst, or in its contents. The cyst-wall itself may become the seat of new growths, and produce secondary cysts, villous, glandular, and other structures: this occurs in many compound ovarian cysts. It may also be the seat of an inflammatory process, terminating in suppuration and granulation, and by this means the cyst frequently becomes obliterated, its contents being either absorbed or discharged externally, and the cavity closing by granulation. Calcification and ossification of the wall may also occur. The contents of cysts undergo various changes, owing to their retention in a closed cavity. The secretions become altered in character, thickened and viscid. Epithelial elements undergo fatty changes, and so give rise to cholesterin crystals. Calcification of the contents is also common.

Cysts may be *simple* or *compound*. A simple cyst consists of a single loculus. A compound or multilocular cyst is one consisting of numerous loculi, which either communicate with one another or remain isolated. Another variety of compound cyst consists of a cyst with endogenous growths, the larger cyst having others growing from its walls. A compound cyst may become a simple one by the destruction of its walls.

Cysts are frequently associated with other growths, hence the terms—"Cystic Sarcoma," "Cystic Cancer," &c. It is especially in those growths which originate in glandular structures, as in the mamma, testicle, and ovary, that this combination is met with. The cystic development may entirely obliterate the structure of the tumor in which it takes place, so that ultimately the latter is converted into a combination of cysts. In other cases large portions of the tumor grow into the cystic cavities. Considerable difficulty is thus not unfrequently caused in determining the nature of the original growth.

CLASSIFICATION.—Cysts may be most conveniently classified, according to their mode of origin, thus:

CLASSIFICATION OF CYSTS.

I. Cysts formed by the accumulation of substances within the cavities of pre-existing structures.

A. RETENTION CYSTS.—Cysts resulting from the retention of normal secretions. These include—

α. *Sebaceous Cysts.*—These are formed by the retention of secretions in the sebaceous glands. Such are comedones and atheromatous tumors.

β. *Mucous Cysts.*—These are formed by the retention of secretions in the glands of mucous membranes.

γ. *Cysts from retention of secretions in other parts*, including, Ranula, from occlusion of the salivary ducts; Encysted Hydrocele, from occlusion of the tubuli testis; cysts in the mammary gland, from obstruction of the lacteal ducts; simple, and some compound cysts of the ovary, from dilatation of the Graafian follicles; and simple cysts of the liver and kidneys.

B. EXUDATION CYSTS.—Cysts resulting from excessive secretion in cavities unprovided with an excretory duct. These include Bursæ, Ganglia, Hydrocele, and many cysts in the broad ligament.

C. EXTRAVASATION CYSTS.—Cysts resulting from extravasation into closed cavities. These include Hæmatocele, and some other forms of sanguineous cysts.

II. Cysts of independent origin.

A. CYSTS FROM SOFTENING OF TISSUES.—These are especially common in new formations, as in enchondroma, lipoma, sarcoma, &c.

B. CYSTS FROM EXPANSION AND FUSION OF SPACES IN CONNECTIVE TISSUE.—These include—

α. *Bursæ*, originating from irritation and exudation into the tissues.

β. *Serous cysts in the neck* (often congenital).

γ. *Many compound ovarian cysts.*[1]

C. CYSTS FORMED AROUND FOREIGN BODIES, EXTRAVASATED BLOOD, AND PARASITES.

D. CONGENITAL CYSTS.—These include many Dermoid cysts. These appear often to be the remains of blighted ova. They contain fatty matters, hair, teeth, bones, &c.

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[1] See Dr. Wilson Fox, on Cystic Tumors of the Ovary, "Med.-Chir. Soc. Trans.," vol. xlvii.

CHAPTER XXVI.

INFLAMMATION.

THE morbid processes which have thus far been described, have been mainly characterized by some alteration in the normal nutrition of the histological elements; either by a diminution in their nutritive activity, as in atrophy and the degenerations; or by an increase, as in hypertrophy and the new formations. In the process of *inflammation*, an alteration in nutrition also plays a prominent part, but changes in the bloodvessels and in the circulation are its essential and most important constituents.[1]

Inflammation is the succession of changes which takes place in a tissue, as the result of some kind of injury, provided that this injury be insufficient immediately to destroy its vitality. With regard to the nature of the injury, it may consist in some *direct* irritation of the tissue, either by mechanical or chemical agents, or by substances conveyed to it by means of the bloodvessels or lymphatics; or in an *indirect* irritation, as in some cases of inflammation of internal organs arising from exposure to cold. In all cases, however, some injurious stimulation of the tissue precedes the occurrence of the local changes which constitute the inflammatory process.

The exact nature of these changes has, for the most part, been ascertained during the past five years, mainly owing to the experimental researches of Professors Cohnheim, Stricker, and Burdon-Sanderson. The method of investigation has consisted in the artificial production of inflammation in the lower animals, and the observation of the process as thus induced. The process comprises—

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[1] With regard to the general pathology of Inflammation, the reader is especially referred to the works of Virchow, Cohnheim, Stricker, and Burdon-Sanderson. The most able article by Professor Sanderson, in the fifth volume of Holmes's System of Surgery, contains an admirable exposition of what is known on the subject.

1st. *Changes in the bloodvessels and circulation.*

2d. *Exudation of liquor sanguinis and migration of white blood-corpuscles;* and

3d. *Alterations in the nutrition of the inflamed tissue.*

It will be well, in the first place, to consider each of these separately, in the order in which they occur, and subsequently to endeavor to point out how far a causal relation exists between them.

I. Changes in the Bloodvessels and Circulation.—Changes in the bloodvessels and circulation, resulting in increased vascularity, have ever been regarded as playing a most important part in inflammation, as upon them principally depend those signs of the process which are most obvious during life. The redness, heat, and swelling, which are so constantly met with in inflamed tissues, are in great measure due to the attendant hyperæmia. The swelling, however, is in most cases dependent rather upon the effusion and cell-growth, than upon the overfulness of the bloodvessels.

These changes in the bloodvessels and circulation are essential constituents of inflammation, both in vascular and in non-vascular tissues. In the latter, which comprise the cornea and cartilage, they take place in the adjacent vessels from which these tissues derive their nutritive supply. The nature of these vascular changes has been studied by the artificial production of inflammation in transparent tissues, in which the circulation can be readily observed; the web and mesentery of the frog, and the wing of the bat, being most convenient for this purpose. The phenomena, as observed in the mesentery of the frog, may be thus briefly described:

The first effect of irritation of the mesentery—mere exposure to the air being sufficient for the purpose—is to cause *dilatation* of the arteries, and subsequently a similar dilatation of the veins. The dilatation of the arteries commences at once, and is not preceded by any contraction. It gradually increases for about twelve hours. This enlargement of the bloodvessels is associated at the commencement of the process with an *acceleration* in the flow of blood; this, however, is soon followed by a considerable *retardation* in the circulation, the vessels still remaining dilated. These alterations in the rapidity of the blood-flow cannot be owing to the increase in the calibre of the vessels, which remain throughout dilated. The relation which subsists between the two phenomena is unknown.

The retardation of the circulation usually commences somewhat suddenly, and is first observable in the veins. The rapidity of the current varies, however, in different vessels; in some—both arteries and veins—it may be more rapid, in others, very slow, oscillating to and fro, or even completely stagnant, these differences occurring perhaps in contiguous vessels, without any obvious cause. The capillaries and small arteries often present at the same time numerous irregular bulgings and contractions; in many parts they may be distinctly aneurismal and varicose.

As the circulation becomes slower, the white blood-corpuscles (leucocytes) accumulate in the veins. Their natural tendency to adhere to the sides of the vessels is increased, so that they may nearly fill the tube. At the same time they exhibit active movements, by means of which they penetrate the walls of the vessels and pass into the surrounding tissues. This phenomenon will be described under the head of "Exudation of Liquor Sanguinis and of White Blood-Corpuscles." The absolute number of white blood-corpuscles may also be increased owing to the irritation of the lymphatic structures in the vicinity of the inflamed tissue.

The red corpuscles also accumulate in the capillaries. They adhere to one another and to the sides of the vessels, and become so closely packed that their outlines can scarcely be distinguished. Increased adhesiveness of the red corpuscles has long been regarded as characteristic of inflammatory blood, by virtue of which they exhibit a greater tendency to cohere in rolls than in health. This was supposed to be due to increased viscidity of the blood owing to an increase in its fibrin. It is doubtful, however, if this be the case—the experiments of Professor Lister tending to show that the aggregation is as marked after the removal of the fibrin, and in non-inflammatory as in inflammatory blood. (See "Causes of Stasis.")

The gradual diminution in the rapidity of the circulation, and the accumulation of the blood-corpuscles in the vessels, are usually ultimately followed by the complete stagnation of the current, constituting the condition long known as *inflammatory stasis*.

II. Exudation of Liquor Sanguinis and Migration of White

Blood-Corpuscles.—Another constituent of the inflammatory process consists in the exudation of liquor sanguinis and migration of white blood-corpuscles.

a. Migration of White Blood-Corpuscles.—The emigration of white blood-corpuscles (leucocytes) through the walls of the bloodvessels was first described, although very incompletely, by Dr. W. Addison in 1842.[1] This observer stated as the result of his researches, that in inflammation these corpuscles adhered to the walls of the vessels and passed through them into the surrounding tissues. In 1846 Dr. Augustus Waller described more fully the same phenomenon, and from his description there can be little doubt that he actually observed the emigration of the corpuscles.[2] Both these observers concluded that the escaped blood-corpuscles became pus-corpuscles. Their observations, however, were but little thought of and were soon forgotten, and it was not until 1867, when similar investigations were instituted quite independently by Professor Cohnheim, of Berlin—to whose minute researches we must ascribe most of our present knowledge on this subject—that the emigration of blood-corpuscles came to occupy an important place in the pathology of inflammation.

The emigration may be observed in the mesentery of a frog which has previously been paralyzed by the subcutaneous injection of curare. The changes in the bloodvessels and in the circulation, and the accumulation of blood-corpuscles in the part, have been already described; it remains only to consider the phenomena of emigration.

The white blood-corpuscles (leucocytes) which have accumulated in large numbers, especially in the veins, remain almost stationary against the walls of the vessel, the blood-current passing by them, although with much diminished velocity. Those immediately adjacent to the wall, gradually sink into it, and pass through it into the surrounding tissue. In doing so they may be observed in the various stages of their passage. At first small button-shaped elevations are seen springing from the outer wall of the vessel. These gradually increase until they assume the form of pear-shaped bodies, which still adhere by their small ends to the vascular wall. Ultimately the small pedicle of protoplasm by which they are attached gives way and the passage is complete, the corpuscle remaining free outside the vessel.

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[1] "Experimental and Practical Researches on Inflammation," Trans. Prov. Med. Association, 1842.

[2] Phil. Magazine, vol. xxix, 1846.

In explanation of this phenomenon, there can be no doubt that the passage of the corpuscles is effected by virtue of their own amœboid activity, by means of which they penetrate the walls of the vessels. The capillaries are now known to consist of protoplasm, and hence the penetration of their contractile walls by the amœboid corpuscles and the subsequent closure of the openings when the transit is completed can be readily understood. The corpuscles having escaped from the vessels into the surrounding tissues, continue to exhibit active movements. They may multiply by division, and thus rapidly increase in number: this will be again referred to when speaking of the origin of pus.

Not only is there an emigration of white blood-corpuscles in inflammation, but the red corpuscles also pass through the walls of the bloodvessels, though in less considerable numbers; and their transit is mainly through the walls of the capillaries. This passage of the red corpuscles takes place in simple mechanical congestion, and it may be observed in the web of a frog in which congestion has been artificially induced by ligature of the femoral vein. (See "Mechanical Hyperæmia.")

β. *Exudation of Liquor Sanguinis.*—Associated with the passage of the blood-corpuscles through the walls of the vessels, is an exudation of the liquor sanguinis. The exuded liquor sanguinis—which constitutes the well-known *inflammatory effusion*—differs from the liquid which transudes as the result of simple mechanical congestion, inasmuch as it usually contains a larger proportion of albumen and fibrin, a proportion which increases with the intensity of the inflammation. It also contains an excess of phosphates and carbonates.

The most characteristic feature of inflammatory effusion is the large number of cell-structures which it contains. These are the direct product of the inflamed tissue, and are in no case generated spontaneously in the effused liquid. Most of them are migrated blood-corpuscles, others are derived from the proliferating elements of the tissue. The quantity and nature of the effusion will thus vary with the tissue inflamed, and with the severity of the inflammatory process. In non-vascular tissues, as cartilage and the cornea, exudation can only occur to a small extent from the neighboring vessels, and hence the effusion is small in quantity. In dense organs—as the liver and kidney, owing to the compactness of the structure, a large amount of effusion is impossible, and what there is, is so intermingled with the structural elements of the organ that it does not appear as an independent material. In the kidney it escapes into the urinary tubes and so appears in the urine. The effusion is most abundant, and constitutes an important *visible* constituent of the inflammatory process, in inflammation of those organs which possess a lax structure and in which the vessels are but little supported—as the lungs, and in tissues which present a free surface—as mucous and serous membranes.

III. ALTERATIONS IN THE NUTRITION OF THE INFLAMED TISSUE.—The remaining constituent of the inflammatory process consists in an alteration in the nutrition of the elements of the inflamed tissue. The nutritive changes, although they may differ according to the structure of the part, are all characterized by an *increase* in the nutritive activity of the cellular elements.

The nature of these nutritive changes has for the most part been ascertained by the investigation of tissues in the lower animals, in which inflammation has been artificially induced. In man, the study of the primary lesions is difficult, owing to the fact that the process can rarely be observed in its earlier stages. These changes will be more fully described when considering inflammations of particular organs and tissues; it will be sufficient in the present place merely to indicate their general characters.

The alteration in nutrition, as already stated, is characterized by an exaltation of the nutritive functions of the cellular elements of the tissues involved in the inflammatory process. This is evidenced by an increase in the activity of those elements which normally exhibit active movements, as the amœboid cells of connective tissue and of the cornea. Cells which under normal circumstances undergo no alterations in form and exhibit no active movements, become active—sending out processes, and undergoing various alterations in shape. This increase in the activity, and variation in the form of the cells, is in most cases followed by enlargement and division of their nuclei and protoplasm, and thus by the formation of new cells.

This increased activity of the cellular elements varies considerably in different tissues, and even in the elements of the same tissue. Some cells exhibit active movements and form new cells much more readily than others. Those tissues, for example, which naturally maintain themselves by the multiplication of their elements, as the epithelial tissues, become active very readily in inflammation, very slight degrees of irritation being sufficient to cause in them rapid cell-proliferation. This is seen in inflammation of mucous membranes, and of the epidermis. In tissues, on the other hand, whose elements normally exhibit no tendency to multiplication, as common connective tissue, cartilage, and bone—active changes are much less readily induced, the cells are much more stable, and multiply with far less facility; and if the inflammation is slight, they may not multiply at all, but simply become enlarged and undergo some alteration in form. Lastly, in the higher tissues, the stability of the elements reaches its maximum, and in nerve-cells no increase of activity can be induced.

Different cells in the same tissue exhibit also different degrees of stability. In common connective tissue and the cornea, for example, the amœboid cells are the least stable, and are the first to multiply. Possibly the age of the cells may influence their tendency to become active, the newer being less stable than the older elements. In all cases, however, the rapidity and extent of the proliferation are in direct proportion to the intensity of the inflammation.

The earliest nutritive change is thus one of cell-proliferation; the subsequent ones are characterized either by impairment of nutrition and the degeneration and death of the newly formed elements, or by the development of these into a permanent tissue. As a rule, it may be said that the new cells are less developed than those from which they originated, more prone to undergo retrogressive changes, and if they form a new tissue, this is inferior in its organization to that of the original structure. This, however, will vary with the tissue involved, and with the intensity of the inflammatory process. The more intense the inflammation, the greater is the rapidity of the cell-proliferation, the more abortive are the young cells, and the less is their tendency to form a permanent tissue.

In connective tissues, these changes in the cells are necessarily accompanied by changes in the intercellular substance. The latter are for the most part characterized by softening. In common connective tissue, the fibres in the first place become succulent and less distinct, and ultimately they are completely destroyed; in cartilage, the matrix softens and liquefies; in bone, the lime-salts are removed, the lamellæ disappear, and the osseous structure becomes converted into medullary tissue. Hence the destructive effects of the inflammatory process.

Having thus briefly described the succession of changes which occurs in the process of inflammation, it remains to consider in what way these result from the injurious stimulation of the tissue, and how far a causal relation subsists between them.[1]

The first apparent change which follows the irritation of the tissue, consists in the dilatation of the bloodvessels, and in an acceleration of the flow of blood. Respecting the cause of this primary vascular phenomenon, recent physiological investigations show that a similar dilatation of the vessels and increase in the activity of the circulation are produced by the excitation of a sensory nerve in those parts in which the nerve originates; and it must be regarded as in the highest degree probable, that the primary vascular phenomena in inflammation are in the same way owing to an injurious impression received by the sensory nerves being reflected by the vaso-motor centre to the vessels. How such an excitation of the nerve produces dilatation of the vessels and increased rapidity of the circulation is, however, unknown.

With regard to the cause of the retardation of the bloodstream, which so quickly succeeds its acceleration, and the ultimate production of stasis, the observations of Professor Lister and Dr. Ryneck tend to show that it consists in some alteration in the walls of the bloodvessels, and not in changes in the blood itself. Dr. Ryneck has shown that stasis may be produced in the web of a frog, in which milk or defibrinated blood has been injected in place of the normal blood; and also that in vessels, the vitality of which has been destroyed or altered by the injection of poisonous metallic substances, no stasis can be produced. These results appear to be conclusive, and show that the retardation and ultimate stagnation of the blood-stream in inflammation, are owing to some alteration in the vital properties of the walls of the bloodvessels with which the circulating blood comes into contact.

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[1] The following conclusions are in the main those arrived at by Professors Stricker and Burdon-Sanderson.

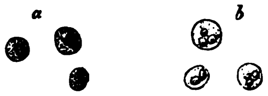
The exudation of liquor sanguinis and of white blood-corpuscles, which take place coincidently with the retardation of the blood-stream, are so closely associated with it, that they must be regarded as being dependent upon the same cause. The walls of the bloodvessels appear to become so altered, that they not only cause stagnation of the circulation, but at the same time permit the liquor sanguinis to transude with abnormal facility, and the white blood-corpuscles to penetrate them.

The remaining constituent of the inflammatory process—the alteration in the nutrition of the inflamed tissue—succeeds the changes in the circulation and the exudation. Respecting the cause of the increased nutritive activity of the cellular elements which characterizes this tissue-change, it is probable that it is for the most part the result of the stimulation of the cells by the liquor sanguinis exuded from the bloodvessels. This conclusion is mainly based upon the well-known experiment of Professor Stricker, which consists in the excision of the cornea of a frog, and its insertion beneath the membrana nictitans of the opposite eye, in which inflammation has been previously induced. The transplanted cornea, when removed in the course of twenty-four hours, exhibits all the inflammatory changes observed in the unexcised cornea of the opposite eye. Hence Stricker concludes that the structural changes in inflammation of the cornea of the frog are owing to the stimulation of its elements by the liquid exuded from the bloodvessels, and are quite independent of nervous influence. That the increased nutritive activity of the elements of the inflamed tissue is, however, in every inflammatory process the result of their stimulation by the exuded liquor sanguinis, cannot be regarded as by any means certain. It is possible that the cellular elements may be stimulated to increased activity by means of impressions conveyed to them through the nervous system, and also that an irritation received by one element may be transmitted to another. This latter hypothesis, however, is not a probable one.

SUPPURATION.—Suppuration, and the formation of abscesses, is a very frequent result of the inflammatory process; it occurs much more frequently, however, in some inflammations than in others. As a rule it may be stated that the more intense the inflammation the more abundant is the formation of pus.

The essential constituents of pus, are cells and a liquid in which they are suspended. The liquid closely resembles the liquor sanguinis. It contains albumen, pyin, chondrin, fatty matters, and inorganic substances. The cells, or *pus-corpuscles* (leucocytes) are indistinguishable from the white corpuscles of the blood. They are spherical, spheroidal, or irregular shaped semi-transparent bodies, from $\frac{1}{2500}$ to $\frac{1}{3500}$ of an inch in diameter, containing a varying number of granules, and usually one or more distinct nuclei. (Fig. 44.) The addition of dilute acetic acid causes the cells to swell up; they become more spherical and transparent, and the nuclei are rendered more apparent. The size of the corpuscles and nuclei, and the number of the granules present manifold variations. Pus-corpuscles, like white blood-corpuscles, lymph-corpuscles, and many other young cell forms—all of which are included under the common term of "*leucocytes*,"—are masses of contractile protoplasm. They possess the power of spontaneous movement, and may undergo continuous alterations in form, or migrate in the tissues. They may also multiply by division.

FIG. 44.



Pus-corpuscles, as seen after death. *a.* Before, *b*, after, the addition of acetic acid. × 400.

The mode of origin of pus has lately been the subject of much controversy. The liquid ingredient proceeds directly or indirectly from the blood; it is the exuded liquor sanguinis: about this there is no dispute. The difference of opinion which exists is respecting the origin of the formed elements. Without discussing the theories which have been advanced by different pathologists, it must be admitted that there are at least *two* sources from which the corpuscles of pus may be derived—one from the *blood*, and the other from the *inflamed tissues.*

It has been seen that, in the process of inflammation, innumerable white blood-corpuscles pass out of the vessels into the surrounding tissues, and as these are indistinguishable from pus-corpuscles, it must be conceded that one mode of origin of pus is from the blood. Further, the white blood-corpuscles may multiply by division, and thus it is probable that by this means the production of pus is greatly increased.

The other source from which the corpuscles of pus are derived is from the cellular elements of the inflamed tissue. These, as has been described, are the seat of active changes in inflammation; they multiply and form new cells, and the more intense the inflammation the more lowly organized are the newly formed elements, and the less is their tendency to form a permanent tissue. Many of these newly formed cells constitute pus-corpuscles; these in this case must be regarded as young elements resulting from the proliferation of the tissue, which are of low vitality, and soon perish. Pus-corpuscles may probably originate in this way, from the proliferation of any tissue with the exception of nerve, either by simple division or by endogenous multiplication.

Although the formed elements of pus may thus be derived both from the blood and from the inflamed tissue, there can be no doubt that the former is their principal source, and that they are in the main migrated blood-corpuscles. In the earlier stages of the inflammatory process they are mostly, if not all, emigrants; but in the later stages it must be admitted that they are also derived from the cells of the inflamed tissue.

Such being the modes of origin of pus, it is evident that the more abundant the escape of blood-corpuscles, and the more active the proliferation of the elements of the inflamed tissue, the greater will be the formation of pus, and hence the greater its tendency to collect so as to form abscesses. It is consequently in those inflammations which are the most intense—provided that the injury is not sufficiently severe to cause instantaneous stasis—that the formation of pus is most abundant. The greater the injury sustained by the walls of the bloodvessels, the more readily will the blood-corpuscles penetrate them, the more rapid will be the cell-proliferation of the tissue, and hence the more abundant the formation of pus. In inflammations of less intensity the escape of blood-corpuscles is less abundant, and the proliferation of the tissue less active, so that pus is not produced in sufficient quantities to cause its collection in the form of an abscess.

When pus is formed in large quantities it exercises a most injurious influence upon the surrounding tissues. The pus-corpuscles appear to be endowed with the power of absorbing the tissues with which they come in contact, or, at all events, of causing their liquefaction. Hence the softening and disintegration of the tissues which constitutes such a destructive element in suppurative inflammations.

Pus which has remained for any length of time in the tissues, undergoes certain changes; thus its elements may undergo fatty metamorphosis, and so be destroyed. If the pus is confined in a closed cavity its liquid portions become absorbed, its cells atrophy, and it may gradually dry up into a caseous mass, which may subsequently become calcified.

VARIETIES OF INFLAMMATION.—Inflammations exhibit certain variations in their characters according to the *severity* of the irritation of the tissue, and the *duration* of its action. Such variations give rise to the division of inflammation into *acute* and *chronic.*

Acute Inflammations.—These result from an irritation of considerable severity, and from one, the action of which is for the most part of short duration. The resulting changes in the tissue run a correspondingly rapid course, and the damage sustained by the bloodvessels and textural elements is correspondingly severe. It is consequently in these inflammations that the vascular phenomena are so pronounced, the exudation of liquor sanguinis and of blood-corpuscles—and hence the formation of pus—so abundant, and the disintegration and softening of the tissue so considerable. The proliferation of the textural elements is also very rapid, and the vitality of the newly formed cells is so impaired that they have little or no tendency to form a permanent tissue. Of these acute inflammations, the process that has been described as resulting from artificial irritation, may be regarded as strictly representative.

Chronic Inflammations.—These differ from the acute, inasmuch as the irritation which causes them is of much less severity, and the duration of its action is also usually much more prolonged. The resulting textural changes, therefore, not only extend over a much greater length of time, but the damage sustained by the tissues is much less severe. The vascular phenomena in these inflammations are consequently not nearly so prominent as in the acute varieties, and the exudation of liquor sanguinis and of blood-corpuscles is for the most part not so abundant. The cell-proliferation also is less rapid and the vitality of the newly formed elements is less impaired, so that they have a much greater tendency to form a permanent tissue. The ultimate tendency of chronic inflammation, therefore, is to produce an increase in the amount of the inflamed tissue. The newly formed tissue, however, is inferior in its organization to the original structure, and more prone to undergo retrogressive changes; and it has a constant tendency to approximate to the normal type. The less severe the inflammation the more permanent and the more highly organized will be the resulting new formation.

Inflammations have also received different names according to the *nature* of the injury upon which they depend. Those inflammations which result from external injuries, mechanical or chemical violence, are called *traumatic*. In those cases in which the injurious stimulation of the tissue is owing to the transmission of infective materials by means of the bloodvessels or lymphatics from some local infecting centre, as in pyæmia and acute miliary tuberculosis, the resulting inflammations are called *infective*. Inflammations in which the nature of the injury is not obvious, are usually called *idiopathic*. Lastly, the nature of the injury may give to the inflammatory process certain peculiarities. The contagion of small-pox, for example, gives rise to inflammation of the skin, constituting the "rash;" that of syphilis to certain inflammations of the skin, mucous membranes, and other tissues; and that of typhoid fever, to inflammation of the intestinal lymphatic structures. In all these and numerous similar cases, the nature of the irritant impresses upon the inflammation certain peculiarities, and in so far as the former is specific, the latter may be called *specific* inflammations.

The pathology of inflammation will be still further elucidated by the study of the process as it occurs in the various tissues and organs. To this end are devoted the next following chapters.

CHAPTER XXVII.

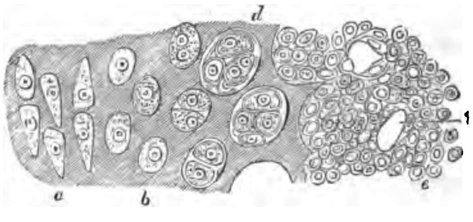
INFLAMMATION OF NON-VASCULAR TISSUES.

INFLAMMATION OF CARTILAGE.

. THE phenomena of inflammation in cartilage have been principally studied by the artificial irritation of the articular cartilages in the lower animals. They consist in changes in the cartilage itself, and in the vessels of the adjacent synovial membrane and bone, from which the cartilage receives its nutritive supply. Respecting the vascular changes, these are such as have been already described as characteristic of inflammation.

In the cartilage itself, the cells enlarge within their capsules, their nuclei become more prominent and their protoplasm more granular. The cells then divide and multiply, so that each cavity contains numerous young cells which ultimately closely resemble pus-corpuscles. At the same time the capsules are destroyed, and the intercellular substance softens and breaks down into a finely granular material. (Fig. 45.) As the intercellular substance becomes destroyed, the newly-formed cells and granular débris escape from the surface of the cartilage, which thus becomes irregular, presenting numerous elevations and depressions: this is ulceration of cartilage. Whilst these changes are taking place in the cartilage itself, the enlarged vessels of the

FIG. 45.



Section of Inflamed Cartilage. *a.* The normal cartilage-cells. *b.* The same enlarged. *d.* Multiplication of cells within their capsules. *e.* Great increase in the number of the young cells, and destruction of the intercellular substance. × 250. (Cornil and Ranvier.)

adjacent synovial membrane and bone extend over its surface and also penetrate its substance; numerous new vessels being formed. This process of softening and ulceration may go on until the cartilage is completely destroyed; but it may become arrested. In the latter case the newly-formed cells, instead of continuing to break down, form a granulation-tissue which subsequently fibrillates, and the lost cartilage thus becomes replaced by fibrous tissue.

When the inflammation is less severe and runs a more chronic course, the cell-proliferation is not so rapid, and the intercellular substance is less destroyed. The newly-formed cells are more highly organized, and many of them form a fibrillated tissue; others undergo retrogressive changes, and thus irregular cavities are produced in the fibrillated cartilage.

INFLAMMATION OF THE CORNEA.

The process of inflammation, as it occurs in the cornea, has been chiefly studied in the frog. After irritation of the frog's cornea, the earliest changes observed consist in the conjunctival epithelium becoming visible, and the appearance amongst the epithelial cells of a few white blood-corpuscles (leucocytes) which have probably escaped from the hyperæmic vessels of the conjunctiva. The cornea-corpuscles and their prolongations then become visible (they are invisible in the healthy cornea, which appears perfectly structureless), and the prolongations are the seat of slight amœboid movements. At a somewhat more advanced stage, the cornea-corpuscles become altered in form, their prolongations become much shorter, they lose their stellate outline, and gradually assume more the appearance of cartilage-cells. The next change observed in the enlarged and rounded corpuscles consists in their proliferation, and in the place of each corpuscle is seen a clump of young, round, amœboid cells, many of which are indistinguishable from the living corpuscles of pus.

Whilst these changes are taking place in the corpuscles of the inflamed cornea, the intercellular substance gradually becomes increasingly opaque, owing to its infiltration with young cellular elements (leucocytes). These are so numerous and increase so rapidly, that they must undoubtedly be regarded as in the main emigrant white blood-corpuscles; although from the changes observed to take place in the earlier stages of the process in the fixed elements of the cornea, it must be admitted that they are partly derived from the proliferation of the cornea-corpuscles. As the number of these young elements increases the consistence of the cornea becomes diminished, until ultimately the tissue breaks down and is destroyed.

The inflammatory process may continue until the whole of the substance of the cornea is destroyed; or it may become arrested. In the latter case more or less thickening and opacity of the cornea will result, owing to the increase in the number of cells and the changes in the intercellular substance.

CHAPTER XXVIII.

INFLAMMATION OF VASCULAR CONNECTIVE TISSUES.

INFLAMMATION OF COMMON CONNECTIVE TISSUES.

COMMON connective tissue is one of the most frequent seats of the inflammatory process, not only the subcutaneous connective tissue, but also the connective tissue of organs and of other parts.

If connective tissue be examined a few hours after the infliction of an injury, it will be found that in place of the fibrillated substance and fixed connective-tissue corpuscles of which it is normally composed, the tissue is infiltrated with amœboid cells (leucocytes), and that the fibrillated intercellular material has become homogeneous and gelatinous in consistence. The number of these cells gradually increases, and the intercellular substance gradually becomes more completely destroyed—being probably consumed by the newly-formed elements; so that ultimately the tissue consists almost entirely of small round cells held together by a very small quantity of soft gelatinous intercellular material.

Respecting the source from which these young elements are derived—*i. e.*, how far they are emigrants, and how far they are the offspring of the cells of the connective tissue, there appears to be little doubt that they are almost entirely emigrants. Although it was formerly supposed that the connective-tissue corpuscles multiplied very rapidly in inflammation, and that the newly-formed cells were entirely the result of their proliferation, the recent investigations of Professors Cohnheim and Stricker show that this view is erroneous; and according to Cohnheim, these corpuscles take no part whatever in the inflammatory process. Professor Stricker, however, has observed them undergoing active movements in the inflamed tongue of the frog, and although he has never seen them divide, he concludes that, like the elements of most other tissues, they probably do so in the later stages of the inflammatory process. The present state of our knowledge respecting inflammation of connective tissue would therefore appear to justify the conclusion that in the early stages of the process all the young cells are emigrants, but that most probably in the later stages many of them are derived from the proliferation of the connective-tissue corpuscles.

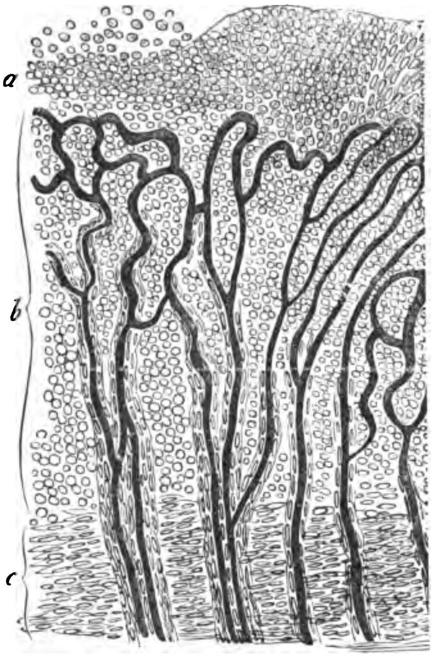
Such being the nature of the changes which more immediately follow injurious stimulation of the connective tissue—the inflammatory process may terminate in *resolution*, in *organization*, or in *suppuration*.

RESOLUTION.—If the injury sustained by the tissue is not severe, the inflammation may gradually subside, the process terminating in *resolution*. In this case the hyperæmia diminishes, the emigration ceases, the young cells undergo fatty matamorphosis and thus become absorbed, possibly also some of them again enter the bloodvessels and lymphatics, and the tissue gradually returns to its normal condition.

ORGANIZATION.—If the inflammatory process does not terminate in resolution, many of the young cells may become more fully developed, and ultimately form a fibrous tissue. This *organization* of the inflammatory formation is seen in the healing of wounds by the "first intention," and also in many of the interstitial inflammations (cirrhoses) of the kidney, liver, and other organs. In order for it to occur, it is necessary that there should be a considerable diminution in the intensity of the inflammation.

The process of organization takes place by the production of new capillaries, and by the development of the young cells into a *granulation-tissue*. This granulation-tissue consists entirely of young cells, which must be regarded as exhibiting a higher stage of development than the primary amœboid cells (leucocytes) of the inflammatory formation. They are spherical masses of protoplasm from $\frac{1}{1800}$ to $\frac{1}{3000}$ of an inch in diameter, containing a distinct round or roundly-oval nucleus, which often only becomes visible after the addition of acetic acid (see Fig. 19); and although they exhibit slight amœboid movements, they are much less active than the emigrant blood-corpuscles. The further organization of the growth takes place by the development of this granulation-tissue into a fibrillated structure. Many of the granulation-cells gradually assume a spindle shape, whilst others disappear, the intercellular substance fibrillates, most of the newly-formed capillaries become obliterated, the remaining cells ultimately become fixed connective-tissue corpuscles, and the tissue is thus converted into a fibrous structure. This new fibrous tissue is characterized by the contraction which it undergoes subsequently to its formation. It is known as *cicatricial tissue.*

FIG. 46.



A granulating surface. *a.* Layer of pus. *b.* Granulation-tissue with loops of bloodvessels. *c.* Commencing development of the granulation-tissue into a fibrillated structure. × 300. (Rindfleisch.)

SUPPURATION.—When the injury sustained by the tissue is so severe, or so prolonged in its action as to prevent the occurrence of resolution or of immediate organization, the young cells infiltrate the tissue in such numbers that they may accumulate so as to constitute pus. The pus may either become collected together within the tissue so as to form an abscess, or it may be continuously discharged from the surface as in a granulating wound. The deleterious influence which the pus exercises upon the tissues with which it comes in contact, causing their destruction and absorption, has already been alluded to.

Organization after Suppuration.—This constitutes what is generally known as healing by *granulation*, or by the *second intention.* It takes place in wounds in which, from the non-apposition of the wounded surfaces, union by the first intention has not been effected; and in other lesions in which the injured tissue presents a free surface communicating with the external air, as in an ulcer. The process of repair after the separation of a dead part (demarcation and separation) is in the same way effected by granulation, as is also the closing of the cavity left after the discharge of the contents of an abscess. In all these, and similar cases, the inflammation and suppuration of the tissue are followed by the formation of granulations, and the subsequent development of these into a fibrillated structure.

The process of organization in these cases is effected in the first place by the development of the young cells in the superficial layers of the inflamed tissue into a granulation-tissue, in the same manner as when there is no suppuration. This granulation-tissue, however, being situated superficially, and coming into contact with the external air, becomes arranged in the form of small papilliform nodules, which are known as *granulations.* This arrangement of the granulation-tissue in the form of granulations, appears to be determined by that of the new capillary bloodvessels which are developed so rapidly in it. These vessels form little vascular loops, and the young cells are arranged round them, so that each loop corresponds with a single granulation. The deeper layers of the granulation-tissue gradually become developed into connective tissue, whilst the cells on the surface of the granulations, together with the liquids exuded from the subjacent vessels, are discharged in the form of pus. (Fig. 46.)

INFLAMMATION OF BONE.

Inflammatory processes in bone give rise for the most part to an increase of medullary tissue and to softening of the osseous structure.

The process takes place in the vascular medullary tissue. This becomes infiltrated with amœboid elements which have emigrated from the hyperæmic vessels. The cells in the medullary spaces and Haversian canals enlarge and multiply; in those which contain fat—the adipose cells—the fat is first removed, and thus a tissue is formed similar to that met with in the medulla during the process of its development. This consists of numerous small round cells, larger cells containing several nuclei closely resembling the "myeloid cells," and a scanty soft intercellular substance. Whilst these changes are taking place in the medullary tissue, the surrounding osseous lamellæ are gradually absorbed, the lime salts are removed, and in this way the medullary spaces and Haversian canals increase in size and ultimately become confluent. There is thus a new formation of medullary tissue at the expense of the compact osseous structure, and the bone becomes exceedingly spongy, soft, and vascular.

As the process proceeds, and the cellular infiltration and formation of medullary tissue increase, the latter may ultimately make its way through the bone and appear as a fungating mass (granulations) beneath the periosteum or the articular cartilage. The extent of pus-formation will depend upon the severity of the inflammatory process. The pus may either accumulate within the cavities formed in the bone, or—as the osseous structure is absorbed and the medullary tissue becomes superficial—make its way to the surface.

The suppuration and rapid growth of tissue, may be the cause of most deleterious ulterior changes in the bone. Owing to the interference with its vascular supply, which results from the pressure exercised by the pus and new tissue as it is confined within the unyielding osseous walls, or to the accumulation of pus beneath the periosteum, smaller or larger portions of the bone may lose their vitality (Necrosis), and the sequestra acting as foreign bodies keep up the inflammatory process. In other cases the bone undergoes a process of molecular disintegration (Caries). As the inflammatory process ceases, or when from its commencement it is less severe, the newly-formed tissue may become organized and form a compact osseous structure, which in some cases is denser than the original bone (Sclerosis).

CHAPTER XXIX.

INFLAMMATION OF BLOODVESSELS AND HEART.

INFLAMMATION OF BLOODVESSELS.

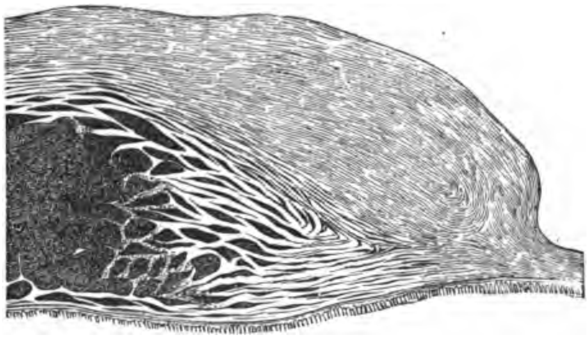
INFLAMMATORY processes, both in the arteries and veins, are not unfrequently associated with the softening and breaking down of coagula which have formed within the vessels, the softened coagulum being the *cause* and not the *result* of the inflammation. A description of these will be found in the chapter on "Thrombosis." Independently of thrombi, it is in the arteries that inflammatory changes are most frequently met with; where they give rise to the various alterations in the walls of the vessel usually known as *Atheroma.*

ATHEROMA.—The changes in the arteries known as *atheromatous*, have their seat in the deeper layers of the inner coat. In the earliest stage of the atheromatous process the fibrous and elastic lamellæ of the inner coat of the vessel become infiltrated with young cells, which are probably partly emigrants, and partly derived from the proliferation of the cells of these structures. As these young cells increase in number they give rise to a swelling beneath the innermost layers of this coat of the artery. (Fig. 47.) This swelling of the intima is very characteristic. It is, in the earlier stage of the process, of a soft flabby consistence, and the lining membrane which is continuous over it can readily be stripped off, leaving the diseased tissue beneath. It thus contrasts strongly with the superficial patches of fatty degeneration which result from the passive metamorphosis of the superficial epithelial and connective-tissue cells of the vessel. (See "Fatty Degeneration of Arteries.")

The termination of the inflammatory process will depend upon its severity. If the process is very acute and the young elements accumulate rapidly, the intercellular substance becomes completely destroyed, and a *pseudo-abscess* is formed beneath the lining membrane of the vessel. The lining membrane may ultimately give way, and the softened matters be carried away by the circulation, and thus is produced the *atheromatous ulcer*. It is these acute arterial changes which are such a fertile cause of the formation of aneurism.

If the process be less acute, and the accumulation of cells less abundant, the destruction of the intercellular substance is much less complete. The young cells undergo fatty degeneration, the more liquid constituents of the degenerated tissue gradually become absorbed, and thus a *caseous mass*, consisting of broken-down fibres and cells, fatty débris and cholesterin, with a varying quantity of the original fibrillated tissue, remains in the deeper layers of the inner coat. (Fig. 47.) This, like other caseous masses, may subsequently calcify and so form a *calcareous plate.*

FIG. 47.



Chronic atheromatous change in an artery. Showing the new tissue in the deeper layers of the inner coat, and the consequent internal bulging of the vessel. In the darker portions the new tissue has become softened and caseous. At the lowest part of the figure are seen a few of the most internal fibres of the middle coat. x 25. (Rindfleisch.)

In the most chronic forms of the atheromatous process there is no softening of the new tissue, but the cells become organized into a fibrillated structure, and thus is produced a *fibroid thickening* of the inner coat of the artery. The organization, however, is rarely complete, more or less fatty débris being usually inclosed in the fibrous stroma.

INFLAMMATION OF THE HEART—ENDOCARDITIS.

Inflammatory processes in the heart, although they sometimes occur in the muscular substance (Myocarditis), are for the most part met with in the endocardium. Here they closely resemble those occurring in the internal arterial coats which constitute atheroma. The endocardium and inner coat are very analogous in their structure, both being almost non-vascular and consisting of fibrous and elastic layers with an internal epithelial covering.

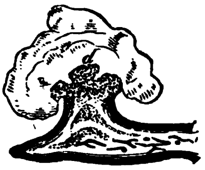
These processes in the endocardium are almost exclusively confined to the *left* cardiac cavities, and in the great majority of cases commence in, and rarely extend beyond, the confines of the aortic and mitral valves and the corresponding orifices. Further—it is those portions of the valves which come into contact in the act of closure and are thus most exposed to friction, which are especially involved, and in which the changes usually commence. Thus, in the aortic valves, it is the convex surfaces of the segments which are most liable to be affected, and not the free edge of the segment, but the little band of tissue which passes from its attached border to the corpus Arantii in the centre; and in the mitral valve, the auricular surface of the segments at a little distance from the attachment of the chordæ tendineæ.

Endocarditis may be either *acute* or *chronic.* In both cases the process has its seat in the subepithelial tissue.

ACUTE ENDOCARDITIS.—If the process be acute, the deeper layers of the endocardium become rapidly infiltrated with young cells, and as these increase in number the intercellular substance becomes softened and destroyed, and thus is produced a soft tissue composed almost entirely of cells such as always results from inflammatory processes in connective tissue. The new tissue as it increases may tilt up the superjacent epithelium, and project in the form of minute granulations and vegetations upon the surface of the softened valve.

The above changes take place in an almost non-vascular tissue, and although there is more or less increase of vascularity in the more external endocardial layers, where the capillaries are more numerous, there is rarely any redness or injection of the endocardium seen after death. Neither is there usually any liquid exuded upon the surface of the granulations. What was formerly regarded as an exuded material, is in the main fibrin, which has been deposited from the blood upon the roughened surface of the valve. This deposition of fibrin frequently occurs in endocarditis; the roughened and abnormal endocardium acts as a foreign body, and so causes a deposition upon its surface. This must not be confounded with the vegetations themselves. (Fig. 48.)

FIG. 48.



Acute endocarditis. A granulation from the mitral valve, showing a fibrinous coagulum upon the surface of the granulation. x 10. (Rindfleisch.)

If the inflammatory process is very acute, the whole of the new tissue may break down, and thus a loss of substance result —an *endocardial ulcer*. This takes place without any accumulation of cells sufficient to form an abscess, the new tissue simply becoming rapidly softened and disintegrating. In rare cases, however, small quantities of pus are found in the deeper endocardial layers. The ulcer is irregularly defined, and its edges are usually swelled and thickened. This ulceration may lead to perforation of the valve, or to a considerable destruction of its substance. Laceration or aneurism may also ensue from the pressure exercised by the blood against the damaged tissue.

If the process be less severe, and the disintegration of the new tissue less complete, the latter becomes incompletely organized into a fibrillated structure, whilst it undergoes, in part, fatty and calcareous degeneration. These changes may result in the adhesion of the valves, either to one another or to the walls of the heart. They always produce permanent thickening, rigidity, and shrinking of their structure. The new tissue may continue to grow after the severity of the process has subsided, and thus are produced the vegetations and papillary excrescences on the valves which are so commonly met with. These consist of a lowly-organized tissue, which quickly undergoes fatty and calcareous changes.

CHRONIC ENDOCARDITIS.—This may be the sequel of acute inflammation, or the process may from its commencement be chronic in its nature. In chronic endocarditis the formation of new cells is much less rapid and abundant than in the acute form ; the intercellular substance consequently becomes much less softened and destroyed, and the new tissue has a much greater tendency to become developed into a fibrillated structure. The extent to which the new tissue becomes organized, however, varies. It may undergo partial retrogressive changes, and thus a loss of substance result (ulceration); or the organization may be more complete and lead to a *fibroid thickening* of the endocardium. In the latter case considerable induration and contraction of the valves or valvular orifice will result. The new tissue frequently forms papillary growths on the valves, which undergo partial fatty and calcareous changes.

CHAPTER XXX.

INFLAMMATION OF LYMPHATIC STRUCTURES.

INFLAMMATORY processes in lymphatic structures usually result from their irritation by substances conveyed to them by the lymphatic vessels. They include—*acute* and *chronic* inflammations, and the inflammation associated with *Typhoid Fever*. Each of these must be considered separately.

ACUTE INFLAMMATION OF LYMPHATIC STRUCTURES.

Examples of acute inflammation of lymphatic structures are furnished by the inflammation of the glands in the axilla from a wound on the hand, of the glands in the groin from gonorrhœa, and of Peyer's and the solitary glands in the intestine from inflammation of the intestinal mucous membrane.

The process consists in a rapid increase in the number of the lymph-corpuscles in the gland, which at the same time becomes exceedingly vascular. This increase is probably due, partly to a hyperplasia of the original cells of the gland, and partly to the migration of blood-corpuscles. The corpuscles not only increase in number, but many of them become much larger in size, and contain several nuclei. The cells of the trabeculæ may also multiply. The gland thus becomes considerably increased in size, soft and pulpy in consistence, and its cortical and medullary parts are no longer distinguishable.

Upon the removal of the source of irritation the process may gradually subside, the new elements undergo disintegration and absorption, and the gland return to its normal condition (Resolution). In other cases the process goes on to suppuration, the trabeculæ are destroyed, many of the cells become disintegrated, and the loculi of the gland become filled with pus. This is usually associated with inflammation and suppuration of the surrounding connective tissue. In the glands of a mucous membrane the process gives rise to what is known as a follicular abscess.

CHRONIC INFLAMMATION OF LYMPHATIC STRUCTURES.

Chronic inflammation of lymphatic structures results from irritations which are less severe and more prolonged in their action than those which give rise to the acute form. The resulting hyperplasia of the elements of the gland is consequently a more continuous one, and the gland becomes more or less permanently increased in size. This hyperplasia takes place not only in the lymph-corpuscles, but subsequently also in the bloodvessels and reticulum, so that all the component structures of the gland become increased. The newly-formed lymph-corpuscles, however, have a great tendency to undergo fatty metamorphoses, and the gland, either in whole or in part, frequently becomes caseous. This is especially the case in the glands of scrofulous subjects. The caseous glands may subsequently soften or become calcified. These retrogressive changes are probably in great measure owing to the obliteration of the bloodvessels, which may ultimately result from the crowding of the cellular elements in the gland. They are often associated with a considerable increase in the reticulum and fibrous structures of the gland.

INFLAMMATION OF LYMPHATIC STRUCTURES IN TYPHOID FEVER.

The inflammatory processes which occur in the lymphatic structures in Typhoid Fever, have their seat in the spleen, in the lymphatic structures of the intestine, and in the mesenteric glands.

The Spleen.—In the spleen the change resembles that which occurs in many of the other acute febrile diseases, although it reaches its maximum in typhoid. The lymphatic elements increase rapidly in number. The organ becomes exceedingly vascular, and often attains two or three times its natural size. Many of the new elements enter the blood, thus causing a slight temporary increase in the number of white blood-corpuscles. As the fever subsides, the hyperæmia diminishes, the hyperplastic process ceases, many of the new elements undergo dinintegration and absorption, the remainder enter the blood, and thus the organ again attains its normal characters and dimensions.

The Intestinal Lymphatic Structures.—It is in the solitary and Peyer's glands that the most characteristic changes take place in typhoid fever. These structures may be involved throughout the whole of the small and large intestine, but in most cases the process is limited to those in the ileum and cæcum; and those glands are always the most affected which are situated the nearest to the ileo-cæcal valve.

The primary change here consists in an increase in the vascularity of the glands, and in a general proliferation of their lymphatic elements. Both Peyer's patches and the solitary glands thus become considerably enlarged and prominent, standing up above the surface of the intestine. They are of a grayish-white or pale reddish color, and of a soft, brain-like consistence. The surrounding mucous membrane is also exceedingly vascular, and is the seat of an acute catarrhal process. This catarrh is more or less general, and usually precedes the swelling of the glands. The new growth, in many parts, rapidly extends beyond the confines of the glands into the immediately surrounding and subjacent tissues, and even in some cases into the muscular coat. It may thus be said to become *heteroplastic.*

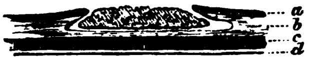
The process now passes into the second stage—that of the death and disintegration of the newly-formed tissue. This may terminate in various ways. The enlarged glands, many of them, subside, the new elements become disintegrated and are absorbed, and the gland thus undergoes a gradual process of resolution. In others, the individual follicles of the gland rupture, discharging their contents externally, and the patches then acquire a peculiar reticulated appearance. The most characteristic termination, however, of the typhoid process, is the separation of the dead tissue as a slough, and the formation of the *typhoid ulcer.*

The process of sloughing and ulceration may, like that of proliferation, take place uniformly throughout the whole gland, in which case the whole mass is thrown off, leaving an ulcerated surface corresponding in size with that of the gland. More commonly, however, in the patches, the sloughing takes place in different portions of the patch, and small irregular losses of substance result, which may gradually extend until they form one large ulcer.

Although, as already stated, the new growth may extend beyond the confines of the glands, this is rarely the case with the ulceration. The heteroplastic growth undergoes resolution, and hence the ulcers have the same configuration as the original glands; those originating from the patches being oval, with their long diameter in the direction of the gut; and those originating in the solitary glands being spherical in shape. In rare cases, when there is much infiltration of the surrounding mucous membrane, the ulceration may extend slightly beyond the confines of the glands.

With the sloughing and disintegration of the new tissue the process of proliferation ceases, and hence there is no induration or thickening of the base or edges of the ulcer. The base is smooth, and is usually formed of the submucous or muscular coat of the intestine. The edges are thin and undermined, and consist of a well-defined fringe of congested mucous membrane. (Fig. 49.) This is best seen when the gut is floated in water.

FIG. 49.



A Typhoid Ulcer of the Intestine (diagrammatic). Showing the undermined edges of the ulcer and the slough still adherent. *a.* Epithelial lining. *b.* Submucous tissue. *c.* Muscular coat. *d.* Peritoneum.

In some cases, however, the sloughing extends deeper through the muscular layer to the subperitoneal tissue, and it may thus cause perforation and peritonitis.

The third stage of the process is that of cicatrization. This takes place by the resolution of the peripheral heteroplastic growth, the approximation and union of the undermined edges with the floor of the ulcer, and the gradual formation from the margin of an epithelial covering. The gland-structure is not regenerated. The resulting cicatrix is slightly depressed, and less vascular than the surrounding mucous membrane. There is no puckering or diminution in the calibre of the gut. In some cases, however, cicatrization does not take place so readily, and the floor of the ulcer becomes the seat of a *secondary* ulceration. This usually takes place after the general disease has run its course, or during a relapse. Profuse hemorrhage and perforation more commonly result from this secondary ulceration than from the primary sloughing of the glands.

The Mesenteric Glands.—The change in the mesenteric glands is probably secondary to that in the intestine. These glands become the seat of an acute hyperplasia, and are enlarged, soft, and vascular. They usually, like many of the glands in the intestine and the spleen, undergo a gradual process of resolution, and thus return to their normal condition. In rare cases, however, the capsule of the gland is destroyed, and the softened matters may escape into the peritoneal cavity, and so cause peritonitis. The enlarged glands may also become caseous, and subsequently calcify.

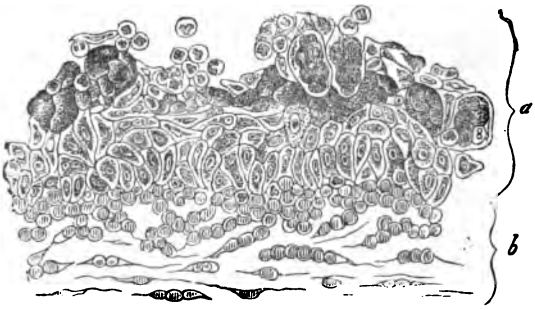
CHAPTER XXXI.

INFLAMMATION OF MUCOUS MEMBRANES.

In mucous membranes inflammatory processes are divided into *catarrhal*, and *croupous* or *fibrinous* inflammations.

Catarrhal Inflammation.—Catarrhal inflammation, or as it is more commonly called *catarrh*, is much the more frequent. In its milder forms this is characterized mainly by an increased secretion of mucus. There is merely an increase in the normal multiplication of the epithelial cells, together with some hyperæmia of the membrane. The small, spherical, newly-formed cells which constitute the mucus-corpuscles are produced with increased rapidity, the liquid which transudes from the blood-vessels is more abundant, and the result is an increased secretion of mucus, rich in corpuscular elements. These young cells are many of them emigrant blood-corpuscles, others are produced within the epithelial cells by endogenous multiplication.

Fig. 50.



Catarrhal Inflammation of the Conjunctiva. *a*. Epithelium. *b*. Subepithelial connective tissue. Showing the proliferation of the epithelium, and the origin of the young elements within the epithelial cells. (Rindfleisch.)

As the proliferation continues many of the epithelial elements become loosened and are discharged with the mucus, and within these groups of bodies may occasionally be seen, which are evidently young mucus-corpuscles. (Fig. 50.) The secretion of the mucous glands is also increased. Owing to these changes, the mucous membrane becomes swelled and abnormally vascular. The increased vascularity is evidenced by redness during life, but after death the blood usually passes out of the vessels, and the membrane may look paler than natural.

If the irritation is more severe, the production of young elements is more rapid, they are smaller and not so well developed, the epithelium loosens and falls off more readily, and the secretion becomes puriform from the great number of corpuscular elements which it contains. Many of these elements are indistinguishable from pus-corpuscles, others are somewhat larger and resemble the corpuscles of normal mucus. Between the corpuscles of mucus and pus there is no line of demarcation, the one passing by insensible gradations into the other. The former are somewhat larger and more regular in shape than the latter, and usually contain only a single nucleus. As the process continues the subepithelial tissue is gradually involved, and becomes infiltrated with young cells. Owing to the loss of epithelium, the surface of the membrane may at the same time become more or less irregular, and present numerous irregular abrasions or ulcers.

These changes in the mucous membrane itself are accompanied by a hyperplasia of the lymphatic structures which it contains. The lymph-follicles become enlarged from the multiplication of their elements. Their contents may soften and form a minute pseudo-abscess, and this bursting gives rise to a small ulcer. These are the follicular ulcers so often seen in catarrhal conditions of the pharynx and intestines. The ulceration in some cases extends beyond the confines of the follicle. The proper glandular structures may also become involved. Their epithelium multiplies, the glands become choked with the epithelial elements, and subsequently atrophy. This is seen in catarrh of the stomach.

The acute process may quickly subside, or it may become chronic. In the latter case the vascularity diminishes, but the multiplication of elements continues both in the epithelial and subepithelial tissue, as does also the increased secretion of mucus (suppuration); the latter becoming thicker and more puriform in character. If this continues for any length of time, the membrane becomes permanently thickened, the subepithelial tissue increases in amount, the lymphatic structures become more or less permanently enlarged, and abrasion or ulceration of the membrane may ultimately result. The enlargement of the lymphatic structures often gives to the membrane a nodular or granular appearance: this is well seen in the pharynx (follicular pharyngitis). In some situations, as the stomach and intestine, the membrane often at the same time becomes deeply pigmented.

CROUPOUS OR FIBRINOUS INFLAMMATION.—This is a more intense form of inflammation than the preceding, and is characterized by the large proportion of fibrin which the exuded liquids contain. The fibrin coagulates within and upon the surface of the membrane. Fibrinous inflammation may be induced artificially by severe irritants, and it occurs in croup, diphtheria, and in some cases of dysentery.

At its commencement the process is simply a severe catarrhal one, consisting in an increase in the vascularity of the membrane, together with an exudation of liquor sanguinis and blood-corpuscles, and proliferation of the epithelial elements. The proportion of fibrin exuded, however, is much greater than in the less severe catarrhal inflammation, and this coagulates more or less completely within and upon the surface of the membrane, inclosing, as it does so, the innumerable newly-formed cell-structures.

Croup.—In croup the exuded fibrin coagulates principally upon the surface of the membrane, where, together with the newly-formed cellular elements, it forms the *false membrane.* This, which can readily be removed from the subjacent epithelial layers, consists of amorphous or finely fibrillated fibrin inclosing innumerable epithelial cells and pus-corpuscles. In consistence it varies considerably, being in some cases firm and tough, in others soft and amorphous. After its removal, the membrane is left partially deprived of its epithelium. If the process subsides the epithelium is reproduced and the part returns to its normal condition; but if it continues a fresh false membrane is formed. As the inflammation extends downwards into the trachea and bronchi, a gradual transition may often be observed from croupous to catarrhal inflammation. In the upper part of the air-passages, where the process is most intense, the exudation is fibrinous, but in passing downwards, where it becomes less severe, the membrane is simply coated with mucus.

Diphtheria.—In diphtheria, the exuded liquids also contain a large proportion of fibrin. The textural change differs, however, from that in croup, inasmuch as the submucous tissue becomes more extensively involved, and the fibrin coagulates not only upon the surface but more especially within the substance of the membrane; and hence it cannot be so readily removed. The pressure exercised by the exuded substances and by the new growth, interferes with the circulation, and so causes the death and sloughing of portions of the membrane; the slough, after it has separated, leaving a considerable loss of substance.

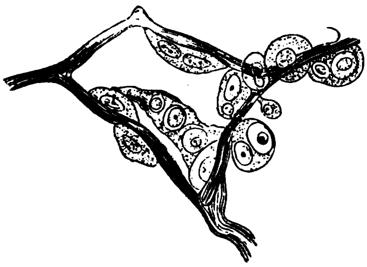
CHAPTER XXXII.

INFLAMMATION OF SEROUS MEMBRANES.

INFLAMMATORY processes in serous membranes vary in their intensity, and in the amount and character of the effusion.

The process commences, as in mucous membranes, with hyperæmia, exudation of liquor sanguinis and migration of blood-corpuscles, together with increased activity of the epithelial elements. The epithelial cells enlarge and become more granular, their nuclei multiply, and thus several new elements are formed within a single cell (endogenous multiplication), from which they subsequently escape, and in this way a number of new cells are produced. (Fig. 51.) The small, round, newly-formed elements, together with some of the older cells, and a large number of emigrant blood-corpuscles, escape with the exuded liquor sanguinis into the serous cavity, where they may continue to exhibit their formative activity.

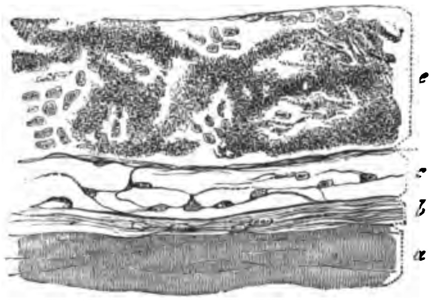
FIG. 51.



Inflamed Epiploon of a Rabbit. Showing the endogenous proliferation of the epithelial cells. x 250. (Cornil and Ranvier.)

Owing to these changes the membrane loses its natural smooth and glistening appearance, and becomes opaque, roughened, and exceedingly vascular. Its surface at the same time becomes covered with a fibrinous layer, and more or less liquid transudes into its cavity. The fibrin, which exudes from the vessels and subsequently coagulates, exists as a soft, elastic, membranous, or reticulated investment, either gluing the two surfaces of the membrane together, or, if they are separated by liquid effusion, forming a slightly adherent layer. (Fig. 52.) The exuded liquid varies considerably in amount, and is always turbid, thus differing from non-inflammatory effusions. It contains flakes and masses of coagulated fibrin and innumerable cell-structures, the latter being in the earliest stages of the process almost entirely emigrants. Similar elements are imbedded in the adherent fibrinous layer.

FIG. 52.



Inflammation of the Diaphragmatic Pleura. Showing the adherent fibrinous layer. *a*. Muscular coat of diaphragm. *b*. Subserous tissue. *c*. Serous membrane. *e*. Fibrinous layer. x 400. (Rindfleisch.)

The nature of the subsequent changes will depend upon the intensity of the inflammation, and upon the amount of liquid exuded into the serous cavity. If the inflammatory process subsides, and the liquid exuded is not sufficient to prevent the two surfaces of the membrane from coming into contact, they grow together and form an adhesion. This constitutes the so-called *adhesive inflammation*. The union is effected by the formation of fibrous tissue. The small round cells imbedded in the fibrinous layer become elongated and spindle-shaped, and form connective-tissue cells; the fibrin fibrillates, and numerous new vessels are formed. Many of the latter atrophy and disappear as the organization becomes complete. The process is thus precisely similar to that which takes place in the union of an incised wound. It is probable also, that in some cases union may take place without the intervention of any fibrinous layer, by the formation and growing together of irregular papillary outgrowths from the subepithelial tissue.

If, however, the inflammatory process is severe, or the surfaces of the membrane are separated by a large quantity of liquid effusion, organization and adhesion cannot be thus readily effected. If a large quantity of liquid exists in the serous cavity, the removal of this becomes necessary before union can take place. If the inflammatory process continues, or its severity is great, union is prevented by the formation of pus. These two conditions must be considered separately.

The existence of a large amount of effusion interferes with the adhesion of the serous surfaces, and before this can be effected the absorption of the liquid becomes necessary. In some cases this occurs very rapidly. More frequently, however, the process is more prolonged, and the subserous connective tissue becomes involved before absorption takes place. When this is the case the subserous tissue becomes infiltrated with young cells, which ultimately form a granulation-tissue beneath the layer of proliferating epithelium. The epithelium itself gradually becomes less abundant, and the exuded fibrin undergoes fatty changes and liquefies. The new granulation-tissue becomes exceedingly vascular, and if the inflammation subsides, it gradually develops into connective tissue, and thus a false membrane is formed rich in vessels, which takes the place of the epithelial layer. As the liquid is absorbed, the two surfaces of the membrane come into contact and grow together, the new vessels becoming gradually obliterated.

If the inflammatory process does not subside, or from its commencement is of considerable severity, it is attended by the formation of large quantities of pus. In this case the emigration of blood-corpuscles is so considerable, and the proliferation of the epithelial cells so rapid, that the young elements exist in large enough numbers to give to the exuded liquid a puriform character. The effusion is then termed *purulent* (empyema). As the submucous tissue becomes involved, and a granulation-tissue is formed, this continues to generate pus, like an ordinary granulating wound. If the pus be removed, the suppuration may gradually cease, the granulation-tissue develop into a fibrous structure, and the union of the serous surfaces thus be effected. The serous membrane becomes greatly thickened, and the new tissue undergoes considerable contraction in the process of its organization.

CHAPTER XXXIII.

INFLAMMATION OF THE LIVER.

INFLAMMATORY processes in the liver are either acute or chronic. *Acute* inflammations, leading to suppuration, are usually infective in their nature, resulting from the transmission of infective materials from lesions in the abdominal organs, or in other parts. The processes are consequently most frequently disseminated and confined to small portions of the hepatic substance. The pus-corpuscles—which usually accumulate so as to form an abscess —are almost entirely emigrants, although recent investigations render it highly probable that they may also originate by the endogenous proliferation of the liver-cells.

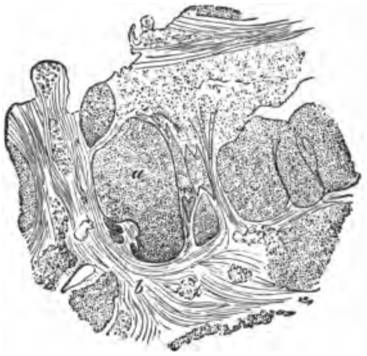
Chronic inflammatory processes in the liver lead to a gradual increase in the fibrous tissue of the organ, and consequently to induration and atrophy of its proper structure. They constitute what is known as *interstitial hepatitis*, or more commonly as *cirrhosis.*

CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver, or interstitial hepatitis, is characterized by a gradual increase in the fibrous tissue of the organ, and by subsequent atrophy of the liver-cells. Whether all changes in the liver in which an increase in the amount of fibrous tissue is the most important structural alteration, are to be regarded as inflammatory in their nature, or whether in some cases they are simple *non-inflammatory* hyperplasias, is uncertain. In either case, however, the ultimate alteration in the hepatic structure is much the same.

The process usually commences in the connective tissue surrounding the smaller branches of the portal vein, and gradually involves that surrounding the larger ones, until ultimately the whole of the interlobular tissue may be increased. The increase may be uniform throughout the organ, or greater in some parts than in others. In the earlier stages, if the growth is rapid, the tissue is exceedingly vascular, being supplied by new vessels derived from the hepatic artery. The cells also are small, round, and exceedingly numerous. This stage, however, is not often observed, as the tissue quickly becomes dense and fibrous, and ultimately may closely resemble cicatricial tissue. (Fig. 53.)

FIG. 53.



Cirrhosis of the Liver. Showing the growth of connective tissue between the hepatic lobules. *a.* Lobules. *b.* New growth of interlobular connective tissue. x 16.

The effect of the new growth is ultimately to cause atrophy of the hepatic cells, and to obstruct the circulation through the portal capillaries and the passage of bile through the bile-ducts. This effect is materially increased by the contraction which the new tissue undergoes. The hepatic cells in the outer zone of the lobules are the first to atrophy; the new tissue often insinuating itself between them, so as to gradually involve the intercellular network. The cells then become smaller and undergo fatty metamorphosis; and ultimately they are completely destroyed. Those in the central parts of the lobule are in the earlier stages but little altered, although they are often stained with bile. As the growth extends, however, these also become annihilated, and the whole lobule is replaced by connective tissue. The cells in the outer part of the lobules are sometimes infiltrated with fat prior to their destruction, the cirrhosis being associated with fatty infiltration.

The portal capillaries also become obliterated by the new tissue,—hence the ascites, hæmatemesis, diarrhœa, enlargement of the spleen, and the other results of portal congestion. Obstruction of the bile-ducts, although it may give rise to staining of the hepatic substance, is rarely such as to interfere with the passage of the bile into the intestine, and hence there is usually but little general jaundice.

Physical Characters.—The liver in the early stages is usually slightly enlarged, the enlargement being almost uniform. The edge is rounded and thickened. If the new tissue is very rapidly produced, the consistence may be somewhat diminished; more commonly, however, the organ is from the first tougher than natural and breaks down less readily under the finger. The characters which it subsequently presents depend upon the distribution of the new tissue. If this is uniform, there will be a gradual and almost uniform diminution in size, and the surface and shape will be but little altered. If on the other hand—as is much more frequently the case—the new tissue is more abundant in some parts than in others, or undergoes irregular contraction, the resulting atrophy will be correspondingly irregular and the surface of the organ will be granular and nodular. In the latter stages, the liver is exceedingly firm, dry, tough, and fibrous. On section, the new tissue is visible to the naked eye surrounding the lobules and in many parts completely replacing them. This gives to the cut-surface a mottled granular appearance, the lobules themselves contrasting with the new interlobular tissue; and this appearance is sometimes increased by fatty infiltration of the cells in the peripheral zone. The capsule also is considerably thickened, and the organ in many parts stained with bile.

Red Atrophy.—This is the condition of the liver which results from long-standing mechanical congestion, such as is caused by disease of the heart. It is characterized by an increase of the interlobular tissue, by atrophy of the liver-cells, and by dilatation and thickening of the hepatic vein. The cells in the centre of the lobule usually atrophy and perish before those at the circumference, and there is an accumulation of black pigment within them and around the radicles of the hepatic vein. The liver in the early stage is increased in size, not from the interstitial growth but from the large amount of blood which it contains. On section it presents a peculiar mottled appearance, the centre of the lobules being of a dark red color, whilst the peripheral portions are of a yellowish-white. This is the true *nutmeg liver*. The subsequent atrophy results partly from the obstruction to the return of blood by the hepatic vein, and partly from the pressure of the interlobular growth.

CHAPTER XXXIV.

INFLAMMATION OF THE KIDNEY.

INFLAMMATORY processes in the kidney present certain variations according as the accompanying structural changes have their principal seat in the tubular epithelium or in the intertubular connective tissue. They comprise, *suppurative*, *interstitial*, and *tubal* nephritis. These must be considered separately.

SUPPURATIVE NEPHRITIS.

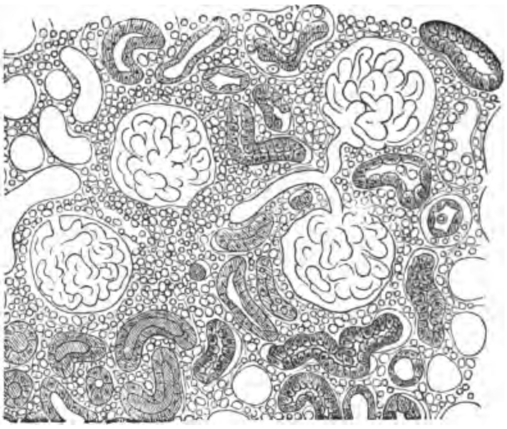
Acute inflammatory processes in the kidney attended by the formation of pus, give rise to *renal abscesses*. Such processes are for the most part associated with inflammation of the pelvis of the kidney (pyelitis), occurring from renal calculus, stricture of the urethra, or enlargement of the prostate. They are also met with in pyæmia. They appear usually to be infective in their nature, and to result from the transmission of infective materials from some primary lesion, this being in some cases an inflamed pelvis—in others—as in pyæmia—a lesion more remote. The resulting suppurative processes—as is always the case in infective inflammations—are consequently disseminated, and numerous abscesses are thus formed in the substance of the organ. These abscesses are often surrounded by a zone of hyperæmic tissue. They may gradually coalesce, and so form one large abscess, occupying the greater part of the kidney substance.

INTERSTITIAL NEPHRITIS.

Interstitial nephritis, or as it is more frequently called, *cirrhosis*, or *granular degeneration* of the kidney, is the most frequent of those morbid processes which constitute Bright's disease. The process is usually chronic in its course, and is characterized, like the corresponding one in the liver, by a gradual increase of the fibrous tissue of the organ, and by the consequent atrophy of the tubal structures.

The first change in the kidney consists in an increase of the connective tissue, between the urine tubes. In the earliest stages of the process the intertubular tissue becomes abnormally vascular and infiltrated with young cells. If the growth is very rapid, these may be exceedingly numerous, and the intercellular substance soft and amorphous. (Fig. 54.) More commonly, however, the process is less acute, and the cells less numerous, and the new tissue quickly becomes developed into a fibrous structure, the bloodvessels at the same time diminishing in number. This is the condition in which it is usually met with. These changes are almost entirely limited to the cortical portion of the kidney, and although here they are more or less general, the new growth is more abundant in some parts than in others, being usually most so around the Malpighian bodies, and in the neighborhood of the capsule, with which it is closely united. In this stage the tubes and their epithelium are unaffected.

FIG. 54.



Interstitial Nephritis, acute form. Showing the cellular infiltration of the intertubular connective tissue. (Rindfleisch.)

In this early stage the kidney is often somewhat increased in size, the capsule usually separates less readily than in health, and the surface of the organ is slightly granular. On section, the cortical substance is in some cases paler, in others redder than natural. The latter is the case when the process is secondary to mechanical congestion. The cut surface also looks obscurely granular. The Malpighian bodies stand out as red points, and the bases of the pyramids and surface of the organ are frequently hyperæmic. The consistence of the organ is usually denser and tougher than natural. This, however, will vary with the character of the new tissue, as will also the increase in size and the irregularity of the surface. If the new tissue is slowly developed and consists principally of fibres, the size will be but little increased, whereas the increase in consistence and the granular condition will be more marked. If, on the other hand, the process is more acute, and the cellular elements predominate, there will be a greater increase in size, the granular character will be slight, and the consistence may be even softer than natural.

The second stage in the process consists in the atrophy of the tubular structures. This is owing to the pressure exercised by the intertubular growth, and to the cicatricial contraction which it undergoes. It is consequently not uniform, but is more marked in some parts than in others. The organ gradually diminishes in size, owing to the atrophy of the cortex, which may ultimately become almost completely destroyed. The bloodvessels become irregularly compressed, in many parts being entirely obliterated: their walls also become thickened. The tubes, like the vessels, are not uniformly affected. Many of them are unaltered, or merely slightly dilated, whilst others become completely destroyed. The epithelium at the same time gradually undergoes retrogressive changes, so that it contains molecular fat. In the latter stages of the disease there is sometimes a proliferation of the epithelium, which accumulates within the tubes. The irregular pressure exercised by the new growth also gives rise to the formation of cysts. These originate partly in the Malpighian capsules, and partly in the urine tubes—the latter becoming irregularly dilated.

In this more advanced stage the kidney is diminished in size. Its surface is more granular, the capsule more thickened and adherent, and it cannot be removed without tearing the kidney substance. The superficial vessels are seen unduly marked in the depressions between the granulations. The cortex is tough and fibrous, of a yellowish-gray or buff color, mottled with yellow streaks and patches; and usually some small cysts are distributed throughout it. Calcareous deposits are also often seen as white streaks between the tubes of the pyramids.

Cirrhosis of the kidney is essentially a chronic disease, and occurs most frequently in middle and advanced life. It appears to be especially associated with the gouty diathesis. It is, however, a frequent result of an irregular life, intemperate habits, and the abuse of alcohol. It is also produced by long-standing mechanical congestion of the organs, such as occurs in heart disease and pregnancy.

When occurring as the result of mechanical congestion, the organs present somewhat different characters from those above described. The interstitial growth in the cortex takes place more uniformly, and hence the granular condition is less marked. The subsequent atrophy also is much less, and the appearance of the organs becomes altered from the large amount of blood which they contain.

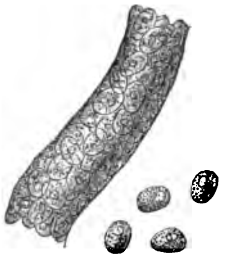
TUBAL NEPHRITIS.

Tubal nephritis, or as it is also called *acute desquamative nephritis*, is another of those morbid processes which constitute Bright's disease, of which it is the most common *acute* variety. The process, which has its seat mainly in the cortex, comprises an increase in the vascularity of the organ, exudation into the urine tubes, and swelling and proliferation of the tubular epithelium.

The earliest change in the organ consists in an increase in its vascularity and exudation into the urine tubes. Many of the capillaries at the same time frequently rupture, and thus there is an escape of blood-corpuscles and of liquor sanguinis into the tubes of the cortex; hence the blood and "blood-casts" in the urine which are so characteristic of the early stage of the disease. This hyperæmia and exudation are followed by swelling and proliferation of the epithelium in the cortical tubes. The epithelial elements become swollen and granular. (Fig. 55.) The granules, which are often so numerous as to occlude the nucleus of the cell, are probably albuminous matters which have been taken up by the protoplasm. They are soluble in acetic acid, and thus differ from molecular fat. This is the condition known as "cloudy swelling" The enlarged and granular epithelial cells then proliferate, and thus a number of new elements are produced. These, together with the older cells, which become loosened, accumulate within and distend the tubes of the cortex, (Fig. 55), and some of them are washed away and appear in the urine as "epithelial casts."

Owing to these changes the organ becomes considerably increased in size and exceedingly vascular. The capsule separates readily, exposing a perfectly smooth, but very vascular surface. The consistence is diminished, the tissue breaking with a soft friable fracture. On section, the increase in the size of the organ is seen to be principally due to the increased thickness of the cortex. This is either of reddish-brown, or of an opaque white or pale buff color; these differences depending upon the relative proportion of blood, and of accumulated epithelial elements. Although in the earliest stage the color is redder than natural, it usually soon becomes pale and opaque. This is owing to the swelling of the epithelial elements, and to their accumulation in the cortical tubes. The blood becomes expressed from the intertubular vessels, and hence the increased vascularity is most evident in the Malpighian corpuscles, beneath the capsule, and in the pyramidal portion of the organ. The Malpighian corpuscles stand out as prominent red points, and the pyramidal cones are of a deep red color, thus contrasting strongly with the pale opaque cortex.

FIG. 55.



Tubal Nephritis—a single urine tube. Showing the accumulation of epithelial elements within the tube. In the few cells which have escaped is seen the granular condition of the cells. × 400.

The termination of the process varies. The increased vascularity and epithelial proliferation may subside, and the newly-formed elements passing away in the urine, the organ gradually return to its normal condition. This is the most common termination when the inflammation is the result of scarlet fever. In other cases the disease continues; and although the vascularity diminishes, the vitality of the epithelial elements has become so much impaired from the inflammatory process, that they undergo retrogressive changes. The cells then continue to come away with the urine adherent to the casts, but instead of presenting the swelled granular appearance as in the earlier stage of the disease, they contain molecular fat. This fat gradually increases in amount as the degeneration proceeds, until it may fill the cells, and ultimately as these are destroyed appear as free molecules and granules on the tube-casts.

This fatty degeneration of the epithelium is attended by corresponding changes in the appearance of the organ. The redness diminishes, and the Malpighian corpuscles are less prominent. The enlarged cortex presents a uniform yellow tinge, studded with minute yellowish-white streaks. This is owing to the presence of fat in the epithelial elements.

This fatty stage, if not far advanced, may undoubtedly pass off. The degenerated cells are carried away by the urine, from those which remain in the tubes the fat is probably partially absorbed, the retrograde process gradually ceases, and the organ may return to nearly its normal size and condition. If, however, the degeneration is considerable and the epithelium becomes extensively destroyed, the disease usually terminates fatally, and after death many of the tubes are found denuded of their epithelium. Whether this loss of the epithelium is ever sufficient of itself to cause a diminution in the bulk of the kidney to such an extent as to give to it a granular surface, appears to be doubtful. Such a result is certainly rare. The organ usually remains large and smooth to the termination of the disease. If, however, the tubular is associated with an intertubular change (cirrhosis), as is frequently the case, atrophy, and consequently a granular surface will readily ensue.

CHAPTER XXXV.

INFLAMMATION OF THE LUNGS.

In the lungs, inflammatory processes comprise the three following varieties: *Croupous*, *catarrhal*, and *interstitial* pneumonia. Of these, the former occurs as an independent affection, whereas the two latter are almost invariably the result of some antecedent pulmonary or bronchial lesion.

CROUPOUS PNEUMONIA.

Croupous, *exudative*, or *lobar* pneumonia, is characterized by intense hyperæmia and by the exudation of a large amount of fibrinous material into the pulmonary tissue. It is termed "croupous" because, like the croupous inflammation of mucous membranes, the exuded liquid contains a large quantity of fibrin. This form of pneumonia almost invariably affects an extensive portion of the lung, hence the term "lobar" which is applied to it. The process is commonly described as consisting of three stages—1st, that of *engorgement*, 2d, that of *red hepatization*, and 3d, that of *gray hepatization*.

In the *first* stage, that of *engorgement*, the lung becomes exceedingly vascular, the changes in the bloodvessels and circulation being such as have been already described as characteristic of inflammation. The organ is of a dark red color, its specific gravity and absolute weight are increased, its elasticity is diminished, it is less crepitant than natural, and pits upon pressure. Its cut surface yields a reddish tenacious liquid.

In the *second* stage, that of *red hepatization*, there is an exudation of liquor sanguinis and migration of blood-corpuscles into the pulmonary tissue. Some of the vessels may also rupture, and thus small extravasations occur. The exuded liquid contains a large quantity of fibrin, which coagulates within the air-vesicles and in the interstices of the interlobular tissue, the coagulum inclosing numerous red and white blood-corpuscles. The lung is now much heavier than in the preceding stage. It contains but little or no air, usually sinks in water, and cannot be artificially inflated. It does not crepitate under the fingers, but is remarkably friable, breaking down readily with a soft granular fracture. The cut surface, which is of a dark red color, presents a granular appearance, the granules being the plugs of fibrin contained in the air-vesicles. Throughout this stage there usually appears to be little or no alteration in the alveolar epithelium or in the inter-alveolar connective tissue.

The *third* stage, that of *gray hepatization*, is characterized by cell-proliferation. The epithelium within the air vesicles multiplies, as do probably the cells in the inter-alveolar tissue, and thus a number of new cells are produced. These new cells, together with the innumerable emigrant blood-corpuscles, are imbedded in the coagulated fibrinous material. The weight, density, and friability of the lung now become even greater than in the preceding stage, although the granular aspect of the cut surface is less marked. The most prominent feature, however, is the alteration which takes place in the color of the organ. This gradually changes from a dark red to a gray or yellowish-white. This is owing partly to the pressure exercised upon the bloodvessels by the exuded substances and newly-formed cells, and partly to the disintegration of the red corpuscles.

Such are the characteristic changes which occur in croupous pneumonia. In those very numerous cases, however, in which the pneumonia is secondary or occurs in debilitated subjects, the changes in the lung present several deviations from those which have been above described. In these secondary pneumonias the exuded substances are less abundant and contain much less fibrin, the cell growth is less active, and consequently the weight of the lung is much less increased. The organ is also softer in consistence and less friable, than when the process occurs in a healthy person. In many cases indeed, slight friability and rottenness of the lung are almost the only evidences which exist of the pneumonic change.

The pneumonic process may terminate in the four following ways:

1st. *In Resolution.*—The gradual return of the lung to its normal condition is much the most frequent termination of croupous pneumonia. The coagulated fibrin liquefies, many of the young cells entangled in it undergo fatty degeneration and disintegration, and thus the inflammatory products become so altered that they can be removed either by absorption or by expectoration. Granular pigment is also mixed with the softened matters and appears in the expectoration. This is probably partly derived from the extravasted blood, and is partly that which normally exists in the interlobular connective tissue. Where this process of liquefaction and disintegration is taking place in the lung, the granular appearance of its cut surface is completely lost. It is of a yellowish-gray color, and a tenacious puriform liquid can be expressed from its substance. As the softened matters become absorbed, the circulation is gradually restored, and the organ ultimately attains its normal characters.

2d. *In Abscess.*—The formation of an abscess is a rare result of simple pneumonia. It may, however, take place if the emigration of blood-corpuscles is very abundant and the cell proliferation is very active. The pus is always mixed with broken-down pulmonary tissue. It is when the pneumonic processes are induced by septic matters and are thus disseminated, as in pyæmia, that the formation of abscess is so liable to occur.

3d. *In Gangrene.*—Still more rare than the preceding is the occurrence of gangrene. Two conditions appear to be principally concerned in bringing about this result: one is the interference with the supply of blood by the extensive formation of coagula in the pulmonary and bronchial vessels, together with considerable hemorrhage into the pulmonary tissue; the other, is the contact of putrid substances with the pneumonic tissue—as the putrid secretion in a dilated bronchus.

4th. *In Phthisis.*—This, which is a very common result of catarrhal pneumonia, is a comparatively rare sequence of croupous inflammation. If, however, the inflammatory products are not removed by absorption and expectoration, they may gradually become caseous, and subsequently by undergoing a process of softening, give rise to the formation of cavities. This is associated with an increase in the interlobular connective tissue (Interstitial Pneumonia).

CATARRHAL PNEUMONIA.

Catarrhal or *lobular* pneumonia differs from the preceding, inasmuch as in it there is but little or no exudation of coagulable material, the process being mainly characterized by proliferation of the alveolar epithelium. It thus somewhat resembles catarrhal inflammation of mucous membranes—hence the term "catarrhal" which is applied to it. This form of pneumonia is almost invariably at its commencement limited to single lobules or to groups of lobules, and does not like the croupous variety involve simultaneously large tracts of pulmonary substance. It is consequently also known as *lobular* pneumonia.

Catarrhal pneumonia very rarely occurs as an independent affection, but is almost always preceded by catarrhal changes in the bronchial mucous membrane, and it appears in many cases to be an extension of the catarrhal process from the latter to the air-vesicles. Collapse of the air-vesicles frequently precedes the pneumonic process, and appears especially to favor its occurrence. It presents acute and chronic varieties.

ACUTE CATARRHAL PNEUMONIA.—Acute catarrhal pneumonia is most common in children and young adults. In children it is much more common than the croupous variety. It is always preceded by capillary bronchitis, and results from an extension of the inflammatory process from the bronchi to the air-vesicles. The collapse of the air-vesicles which is so frequently caused by the bronchitis, appears greatly to favor the occurrence of this pneumonic change.

The catarrhal process consists in enlargement and proliferation of the alveolar epithelium, together with more or less hyperæmia and serous exudation from the surrounding bloodvessels. The newly-formed elements are large and spheroidal, usually containing one, sometimes two or three, bright and comparatively small nuclei, and in a more advanced stage, molecular fat. As they increase in number they completely fill and distend the alveolar cavity. On section of the lung, the pneumonic lobules are seen scattered through its substance, as small reddish-gray nodules, with a smooth and slightly granular surface. The immediately surrounding lung-tissue is usually hyperæmic and œdematous, and frequently collapsed. In a more advanced stage the newly-formed elements undergo fatty metamorphosis, and the nodules change from a reddish to a yellowish-gray color. As the process continues, the numerous small pneumonic nodules gradually coalesce, and thus there may be formed large masses of reddish-gray friable consolidation. Resolution and the return of the lung to its normal condition may occur in this stage. Such a result, however, owing to the abundant cell-growth, is far less common than in croupous pneumonia. Very frequently the fatty metamorphosis of the new elements is incomplete, consequently they do not become absorbed, but the pneumonic masses become caseous, and the disease thus passes into the chronic stage.

CHRONIC CATARRHAL PNEUMONIA AND PULMONARY PHTHISIS.—Chronic catarrhal pneumonia plays the most important part in the production of pulmonary phthisis. It may be the result of the above-described acute process, or, as is much more frequently the case, it is from the commencement a chronic change. The process, like the acute, is almost invariably preceded by catarrh of the bronchial mucous membrane, and is characterized mainly by the development and accumulation of cellular elements within the pulmonary lobules. The pneumonic nodules, however, very rarely undergo complete fatty metamorphosis and resolution, but gradually become *caseous*, and the resulting caseous masses become themselves the seat of subsequent changes, whilst at the same time they cause changes in the pulmonary tissue.

The question of the nature of the morbid processes which occur in the lungs in pulmonary phthisis has already been briefly alluded to, when speaking of tubercle in these organs (see "Tubercle in the Lungs"). It was there seen that these processes are for the most part pneumonic in their character, the pneumonic products subsequently becoming caseous, and that in the comparatively few cases in which tubercle is met with, it is much more frequently to be regarded as a secondary than as a primary growth. Both the croupous and catarrhal forms of pneumonia may, by terminating in caseation, be causes of phthisis. In croupous pneumonia, however, such a result is rare, whilst in the catarrhal forms it is exceedingly frequent. Acute catarrhal pneumonia, as already stated, often terminates in caseation, and it may thus be a cause of phthisis. Many of the cases of acute phthisis in children and young adults, such as occur after measles and whooping-cough, and are usually known as tuberculosis, are of this nature. It is catarrhal pneumonia, however, in its chronic form, that is the most frequent cause of pulmonary phthisis, because in it caseation of the pneumonic products is the rule, whereas in the acute form and in croupous pneumonia this is only an occasional sequence.

Chronic catarrhal pneumonia, like the acute variety, is probably invariably secondary to catarrhal processes in the bronchial mucous membrane, to which the proliferation of the alveolar epithelium and its accumulation within the alveolar cavities must be regarded as owing. The extreme frequency with which the development of phthisis is preceded by successive attacks of bronchial catarrh is a well-established clinical fact, the important bearing of which upon the successful treatment of the disease it would be difficult to over-estimate. The first effect of such catarrhal processes in the bronchi is to cause an increased secretion of mucus. This is ultimately followed by thickening of the mucous membrane, and by an increase in the peribronchial connective tissue with more or less irregular contraction and dilatation of these tubes. The changes in the bronchi precede and probably determine the accumulation and subsequent caseation of cellular elements within the pulmonary lobules, which constitute the primary anatomical feature of the very great majority of cases of phthisis.

The development of the catarrhal pneumonia which succeeds the changes in the bronchi, appears to be owing partly to the extension of the catarrhal process from the terminal bronchioles to the air-vesicles, and partly to the collapse of the air-vesicles which so frequently results from the accumulation of mucus within the smaller tubes. The occurrence of collapse especially favors the proliferation of the alveolar epithelium. This is probably owing to the removal of the normal pressure from the alveolar bloodvessels, which consequently become more or less enlarged and the blood tends to stagnate within them. In whichever of these ways the epithelial proliferation is induced, the newly-formed cells gradually accumulate within the alveoli, which, together with the terminal bronchioles, thus ultimately become filled with cellular elements. Probably in many cases the accumulation is increased by the inhalation of some of the bronchial secretion. As the process continues the isolated pneumonic nodules become confluent, and so form large masses of consolidation, until ultimately the whole lung may become involved.

These accumulations of cells almost invariably atrophy and undergo partial fatty metamorphosis, this appearing to result from the pressure to which they are subjected within the alveoli. The gray pneumonic nodules and larger masses of consolidation thus become yellow and *caseous*. The changes which subsequently take place in the caseous masses vary: they may either gradually dry up and become calcified, or undergo a process of disintegration and liquefaction. Which of these changes occurs is probably partly determined by the extent of the accumulation within the alveoli. If this is not sufficient to exercise an injurious influence upon the alveolar walls and their vessels, the caseous masses may gradually atrophy, dry up, and become infiltrated with calcareous particles, and the resulting cretaceous mass, ultimately becoming encapsuled by fibrous tissue, undergoes no further change. If, however, the accumulation within the alveoli is so great as to cause considerable pressure upon the surrounding parts, and thus to interfere materially with the circulation, the caseous masses become softened and disintegrated, as do also the alveolar walls and the adjacent structures, and in this way a cavity is produced.

Whilst these processes are progressing within the alveoli, changes of a different nature are taking place in the pulmonary tissue, the extent and nature of which principally depend upon the chronicity of the catarrhal process. These changes mainly consist in a hyperplasia of the pulmonary connective tissue (interstitial pneumonia). This tissue increases around the small bronchi and bloodvessels, around the alveolar walls, which thus become thickened, and it also forms capsules around the pneumonic masses which have dried up or become calcified. The more chronic the catarrhal process and the resulting caseation, the more abundant is the growth of connective tissue, so that in the most chronic cases of phthisis the pulmonary substance may be almost completely replaced by a dense fibroid growth. The new connective tissue, which continues to contract after its formation, causes diminution in the size of the lung, retraction of the thoracic walls, and dilatation of the bronchi. The bronchi often become dilated in this way to such an extent as to form cavities, and thus many of the cavities in phthisical lungs are produced.

The adenoid tissue around the bloodvessels and in the neighborhood of the alveoli and small bronchi also increases, and thus large tracts of this tissue are found associated with the fibrous growth. There is frequently at the same time a development of true tubercle, this, as already stated, being probably in most cases secondary to the caseous pneumonic products (see "Tubercle in the Lungs"). The tubercles can only be recognized as such in the earlier stages of their development; when they have become completely caseous, they cannot be distinguished from the other caseous products. It is important not to confound all the minute nodules met with in phthisical lungs with true tubercle. Many of these are the little gray masses of pneumonic consolidation; others, again, are transversely-divided bronchi with thickened walls and caseous contents.

INTERSTITIAL PNEUMONIA.

Interstitial pneumonia is a chronic process, and is characterized by an increase in the connective tissue of the lung, which is precisely similar to that which occurs in the liver and kidney, where it constitutes cirrhosis. The process in the lung, however, differs from that in these organs inasmuch as it rarely, if ever, occurs as a primary and independent affection. The newly-formed tissue, like all other inflammatory growths of connective tissue, is at first rich in cells and bloodvessels, but it subsequently becomes converted into a dense, fibrous, contracting structure.

An increased growth of connective tissue in the lung is almost invariably preceded by some other inflammatory process either in the lungs or bronchi. It may be stated generally that all chronic pneumonic or bronchitic processes are attended by an increase in the pulmonary connective tissue. In phthisis, as has been seen, this tissue increases; and the more chronic the disease the greater is the increase. In the most chronic cases the pulmonary tissue may be almost completely replaced by a dense fibrous growth, which, from its contractile properties, causes a diminution in the size of the lung and dilatation of the bronchi: such a condition has been described as "fibroid phthisis." Simple chronic croupous pneumonia also leads to an interstitial growth. In chronic pleurisy, again, the connective tissue in the superficial parts of the lung is considerably increased, dense septa of it passing inwards from the subpleural tissue. Chronic bronchitis is also frequently attended by an increase of the connective tissue, which occurs at first around the bronchi, but subsequently may involve other portions of the lungs. In that form of it which results from the inhalation of irritating particles, and terminates in ulceration of the bronchi, catarrhal pneumonia, and destruction of the lungs—the so-called "Colliers'" and "Knife-grinders' Phthisis," &c.—the fibrous growth is very considerable (see "Pigmentation of the Lungs").

Whether interstitial pneumonia ever occurs as a primary and independent affection is extremely doubtful, although such a condition has been described under the name of "*cirrhosis of the lung.*"

CHAPTER XXXVI.

INFLAMMATION OF THE BRAIN AND SPINAL CORD.

INFLAMMATORY processes in the nervous centres are probably much less frequent than was formerly supposed. Many of those morbid changes in the brain and spinal cord which are attended by softening, and which were at one time regarded as the result of inflammation, are now known to owe their origin to simple interference with the vascular supply, such as results from embolism, thrombosis, or degenerative changes in the walls of the bloodvessels (see "Fatty Degeneration of Brain").

The irritation which determines the occurrence of inflammation in the brain or cord, is most frequently some external violence—a blow, simple concussion, or fracture of the osseous framework. In other cases it is diseased bone, as in the inflammation of the brain which so often results from disease of the petrous portion of the temporal bone.

The resulting inflammatory process is almost invariably limited to small portions of the cerebral or spinal substance. It is attended by the formation of pus, and usually leads to the formation of an *abscess.* The earliest change consists in intense and localized hyperæmia, which is frequently attended by rupture and minute extravasations. The nervous tissue then becomes infiltrated with young cells and considerably softened, and presents a uniform red or mottled color. The nerve-fibres become disintegrated, the nerve-cells undergo fatty degeneration, and may thus form the "inflammatory," or "exudation corpuscles" (see Fig. 3, *b*). In most cases the accumulation of young cells is sufficient to give rise to the formation of an abscess, and a yellowish or reddish puriform liquid takes the place of the original softened mass. The tissue surrounding the abscess is also hyperæmic, softened, and infiltrated with cells. The abscess may gradually extend, or it may become limited and encapsuled by the formation of connective tissue from the neuroglia. When thus encapsuled, it may gradually dry up into a caseous or calcareous mass, or the absorption may be more complete so as to leave little more than a cicatrix. Respecting the source from which the young cells are derived,—they are probably almost entirely emigrants, although they are possibly also partly the offspring of the cells of the neuroglia. The nerve-cells themselves appear to undergo no active changes in inflammation.

In addition to these localized and suppurative inflammatory processes, changes of a much more chronic nature, and much wider and more general in their distribution—which are probably to be regarded as inflammatory—are met with in the nervous centres. These are characterized by a gradual increase in the connective tissue (neuroglia) and by atrophy and disintegration of the proper nervous elements.

INFLAMMATORY SOFTENING.—Conditions of softening of the cerebral or spinal substance resulting from inflammation, other than those which have been above described, probably rarely, if ever, occur as *primary* lesions. Most varieties of softening, which were formerly described as inflammatory—either from the red color of the softened tissue, or from the acuteness of the process—result, as already stated, from embolism (see "Embolism in the Brain"), or are simply passive degenerative changes, in which more or less extravasation of blood has taken place into the softened tissue (see "Fatty Degeneration of Brain"). Inflammation and consequent softening of the nervous tissue, however, by no means unfrequently occurs as a *secondary* process. It takes place especially around clots of blood or other morbid products within the brain or spinal cord, and results from the injurious influence which these substances exercise upon the immediately adjacent structures. Such a result is not unfrequent in cases of cerebral hemorrhage. The nerve-tissue immediately surrounding the clot becomes the seat of an inflammatory process, and it is found after death softened, hyperæmic, and infiltrated with young cells. Inflammation of the superficial portions of the cerebral and spinal substance also occurs as the result of meningitis.

CHAPTER XXXVII.

CHANGES IN THE BLOOD AND CIRCULATION.

HYPERÆMIA.

HYPERÆMIA or congestion is excess of blood in the more or less dilated vessels of a part. Whatever increases the pressure of the blood, or diminishes the resistance of the vessels, may be a cause of hyperæmia. Hyperæmia, is *active* or *arterial*, and *mechanical* or *venous*. These two varieties must be considered separately.

ACTIVE HYPERÆMIA.

Active hyperæmia is an excess of blood in the arteries of a part, with, in most cases, an acceleration of the flow.

CAUSES.—The causes of active hyperæmia may be divided into those which increase blood-pressure, and those which diminish arterial resistance.

1. *Increased Blood-pressure.*—This occurs most commonly from interruption of the main current of blood in any particular part, owing to which increased pressure is thrown upon the collateral vessels. These vessels thus become dilated, the amount of blood in them is increased, and the flow is accelerated. This, which is known as *collateral hyperæmia*, is seen after the obstruction of the main current from any cause, as from the ligature of the vessel, or from its occlusion by a thrombus or embolus (see "Embolism").

General obstruction in the capillaries of a part will in the same way cause a compensatory hyperæmia This is exemplified by the application of external cold causing contraction of the superficial capillaries and congestion of internal organs; and by obstruction of the capillaries in one part of an organ causing hyperæmia of the parts adjacent.

2. *Diminished Arterial Resistance.*—This is much the most frequent cause of active hyperæmia. It may arise from—

a. Relaxation or paralysis of the wall of the vessel.—The relaxation of the muscular coat of an artery and the consequent dilatation of the vessel, may be owing either to the direct paralysis of the vaso-motor nerve supplying it, or to the irritation of a sensory nerve. The effects of direct paralysis of the vaso-motor nerves are seen in the active congestion of the head and neck which follows pressure upon the sympathetic in the neck, as by an aneurism ; and in the unilateral congestion which results from experimental sections or disease of one-half of the spinal cord. Some emotional conditions also are attended by paralysis of the vascular nerves and consequently by active hyperæmia: this is seen in blushing. Certain substances again taken internally produce vaso-motor paralysis, as the nitrite of amyl, alcohol, tobacco, &c.

Relaxation and dilatation of the arteries is also produced by the irritation of a sensory nerve, in those parts in which the nerve originates. Active hyperæmia from this cause has already been described when speaking of it as the earliest change in the process of inflammation. The irritation received by the sensory nerves is reflected by the vaso-motor centre to the bloodvessels, and causes dilatation of the vessels and increased rapidity of flow. In inflammation, this irritation is so severe as to cause not only dilatation of the vessel, but also changes in its walls, which ultimately lead to the retardation of the blood-flow and exudation of liquor sanguinis and blood-corpuscles. If the irritation be less intense or less prolonged in its action it produces simply dilatation of the vessels and increased rapidity of flow—*i. e.*, active hyperæmia. This is seen in the congestion of the skin which results from friction, heat, and many irritating substances; in the priapism that sometimes results from the passage of a catheter; and in many similar conditions.

β. Sudden removal of external pressure.—The sudden removal of external pressure from vessels is followed by their dilatation, and consequent hyperæmia. As examples of hyperæmia from this cause may be mentioned that which results from dry cupping, and from the sudden removal of ascitic fluid, and of the fluid from a hydrocele.

γ. Atony of the walls of the vessels from mal-nutrition.—This is a much less important cause of hyperæmia. Fatty degeneration of the muscular and internal coats of the smaller arteries may, however, in some cases, lead to their dilatation, and thus be a cause of active hyperæmia.

RESULTS.—The results of active hyperæmia are principally such as might be expected to follow from an increase in the amount of the arterial blood, and in the rapidity of its flow, in any particular organ or tissue. There is increased redness and pulsation, a sensation of throbbing being often experienced by the patient. There is also an increase in bulk. The temperature at the same time undergoes a marked elevation; as much as 10° Fahr. has been observed after injuries of the spine, and experimental sections of the sympathetic. Serous effusions, hemorrhage, and thrombosis—so common as results of mechanical hyperæmia—are rarely met with. If the hyperæmia is of long duration, the small arteries become permanently enlarged, and their walls thickened. Function may, or may not, be interfered with. It is in the nervous centres that functional changes are most frequently met with. They include great excitability, paræsthesiæ of sight and hearing, convulsions, &c.

MECHANICAL HYPERÆMIA.

In mechanical hyperæmia the excess of blood is principally in the veins, and the flow, instead of being accelerated, is retarded.

CAUSES.—The causes of mechanical hyperæmia are such as interfere with the return of the blood by the veins, either by directly impeding its exit from any vein or system of veins, or by diminishing the normal circulating forces. They are:

1. *A Direct Impediment to the Return of Blood by the Veins.*—This is the most fertile cause of mechanical hyperæmia. Any obstruction to the return of blood by the veins is followed by distension and impeded flow behind the obstruction. The congestion of the abdominal viscera which results from the obstruction to the portal circulation in cirrhosis of the liver, and of the lung in mitral constriction and regurgitation; that of the systemic circulation in insufficiency of the tricuspid valve; and that of the lower extremities from the pressure of the gravid uterus on the iliac veins, are a few of the numerous familiar examples of mechanical hyperæmia from this cause.

2. *Gravitation.*—This becomes an important auxiliary in the production of hyperæmia in disease. especially when it is associated with diminished cardiac power. The effect of gravitation in determining congestion of the most dependent parts is exemplified in chronic exhaustive and in many of the acute febrile diseases, in which the nutrition generally becomes impaired, the heart's power weakened, and in which the patient is unable frequently to change his position. The integuments of the back, and the posterior portions of the lungs, are the parts which are thus most frequently affected.

3. *Increased Local Resistance.*—This results from diseased conditions of the arterial walls, owing to which they either lose their elasticity and contractility, and thus their power of equalizing and regulating the blood-flow, or become considerably enlarged. In either case the circulation will be impeded, and accumulation of blood and retardation of flow take place in the veins beyond. Such conditions arise from atheromatous, fatty, and calcareous changes in the arterial walls, and are most common in advanced life. The part they play in the production of senile gangrene has been already alluded to (see "Senile Gangrene").

4. *Diminished Cardiac Power.*—This is one of the most important causes of mechanical hyperæmia, especially when it is associated with any of the preceding ones. The motor power of the heart becomes impaired in many of the chronic exhausting diseases, also in the acute febrile diseases, as in typhus and typhoid fever, and in all those conditions of degeneration and softening of its structure which lead to the dilatation of its cavities. In whichever of these ways the *vis a tergo* is diminished it will tend to produce venous hyperæmia.

RESULTS.—Long-continued mechanical hyperæmia leads to impairment of vitality and function. The tissues gradually atrophy and undergo retrogressive changes, although from the amount of serosity and blood which they contain their size and absolute weight may be increased. Their temperature becomes lowered. The most important results, however, of this variety of hyperæmia are the *transudation of serum*, *hemorrhage*, *thrombosis*, and *gangrene.*

The *transudation of serum* into the surrounding tissues, constituting œdema and dropsical effusion, results most frequently from direct venous obstruction. Its occurrence is greatly favored by the stretching of the walls of the vessels, and by the damage which their structure sustains. The transuded serum usually differs from blood-serum in being of lower specific gravity, and in containing more water and less of the solid constituents. The greater the pressure, the more nearly does the transuded liquid resemble the liquor sanguinis, and the greater is the amount of albumen which it contains. If the pressure be very great it may yield a fibrinous coagulum.

Another result of mechanical hyperæmia is *hemorrhage*. This usually occurs only when the obstruction to the venous current is very great. Those vessels which are the least supported are the first to give way. The hemorrhage into the stomach in cirrhosis of the liver, and into the lung in mitral disease, are familiar examples of hemorrhage from this cause.

Not only does blood escape from the vessels by rupture of their walls in mechanical hyperæmia, but the red blood-corpuscles pass through the walls of the capillaries into the surrounding tissues without rupture taking place. This passage of the red corpuscles through the capillary walls, which was discovered by Cohnheim, may be observed in the web of the frog's foot after ligature of the femoral vein. The corpuscles in passing through the vessel become constricted in their centre, so as to assume an hour-glass shape. This emigration only occurs when the obstruction is considerable.

The occurrence of *thrombosis*, as the result of mechanical obstruction, will be described in the following chapter.

Gangrene only occurs from mechanical hyperæmia when the obstruction is very general and complete. It has been already described under the head of "Gangrene."

Post-mortem Appearances of Hyperæmia.—The post-mortem appearances presented by hyperæmic organs and tissues vary considerably. Very frequently, parts which were hyperæmic during life show no signs of it after death. If the blood does not coagulate rapidly it passes on into the veins, and thus the recognition of arterial and capillary hyperæmia very often becomes impossible. The effect of gravitation must also be taken into account in estimating hyperæmia. After death the blood naturally gravitates to the most dependent parts: this is seen in the post-mortem congestion of the posterior portions of the lungs, and of the most dependent portions of the various coils of the intestine. The uniform redness of post-mortem staining, again, must not be confounded with the redness of hyperæmia. In capillary and arterial hyperæmia, the color is red, and the injection often presents the appearance of a capilliform network. If very intense, it may to the naked eye appear uniform, but a lens will always discover its capillary nature. When the veins are the seat of the hyperæmia the injection is called ramiform, and the color is dark blue.

The anatomical peculiarities in the distribution of the vessels will, however, materially affect the appearance of the hyperæmia. In the intestines it is often punctiform, being situated in the vessels of the villi; so also in the kidney, when its seat is the Malpighian corpuscles. A punctiform appearance may also be produced by minute extravasations of blood. If the hyperæmia is of long standing, the tissue becomes pigmented. This is often well seen in the stomach and intestines, also in the lungs.

CHAPTER XXXVIII.

THROMBOSIS.

THROMBOSIS is a coagulation of the blood within the vessels during life, owing to changes in the walls of the vessel itself, or to impeded blood-flow. The coagulum is called a *thrombus.* It may form in the heart, in the arteries, in the capillaries, or in the veins. It is much the most common in the last-named vessels.

Thrombi must be distinguished from the coagula that form after death, and also from those formed in the last moments of life, which are so commonly found in the cardiac cavities. Post-mortem coagula are soft, and are often divisible into two layers, colored and uncolored; they do not adhere to the walls of the vessel, and rarely completely fill its cavity. The clots formed in the heart just before death constitute a connecting link between post-mortem coagula and thrombi. They are usually more or less decolorized. and are firmer in consistence, and more fibrinous than post-mortem clots. They are not firmly adherent to the cardiac walls, but are often so entangled amongst the columnæ carneæ, chordæ tendineæ, and papillary muscles, that they cannot be quite readily separated. They appear to be the result of the mechanical defibrination of the blood by the cardiac contractions just before death; the contractions not being sufficiently strong to empty the cavities, some of the blood remains behind, and becomes "whipped up" and defibrinated. These clots are most common in the right cardiac cavities, and they often extend some way into the pulmonary artery and aorta, from which, however, they can very readily be removed. They are met with most frequently in those cases in which death has taken place slowly, and in which there has been a gradual loss of power in the cardiac contractions. The proportion of fibrin in the blood will also materially influence the extent of their formation.

A *thrombus*, or ante mortem clot, is firmer, drier, and more fibrinous than either of the preceding, and it is adherent to the walls of the vessel. Its characters, however, vary with its age, and with the circumstances under which it originates. When freshly-formed it is of a dark-red color, and soft gelatinous consistence, closely resembling the post-mortem clot. It gradually becomes paler, drier, less elastic, and more friable. If it is rapidly produced—the circulation being suddenly arrested in a portion of a vessel, as by the application of a ligature—the coagulum at once completely fills the vessel, and as it becomes firmer it maintains a more or less uniform structure. Thrombi, however, which have undergone a slow and gradual formation, are rarely thus uniform in structure, but are made up of numerous concentric layers, and so present a stratified appearance. This is owing to the coagulation taking place gradually upon the inner surface of the vessel, and to the white corpuscles adhering to the successive layers of coagulum. These corpuscles, from the property which characterizes them of adhering to one another and to the sides of the vessels, and especially to any porous substances with which they may come in contact, cohere with the layers of fibrin as they are deposited; so that ultimately the thrombus is made up of layers of fibrin and white corpuscles more or less alternately and concentrically arranged. This gives to it a stratified appearance.

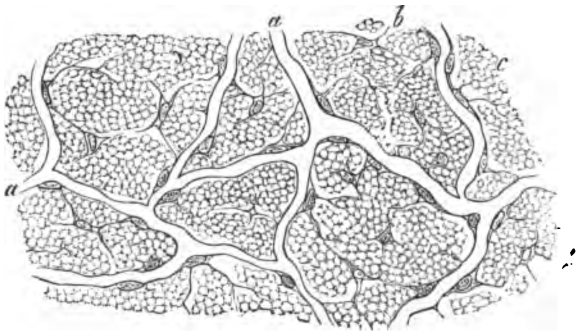
The thrombus may completely or only partially fill the cavity of the vessel. In most cases, however, when coagulation has commenced, it proceeds until the vessel is obstructed, and when once this has occurred, the formation of the thrombus continues to extend in the course of the vessel until it meets with a current of blood strong enough to arrest its progress. Its ultimate extent will thus mainly depend upon the vessel in which it is formed, upon the size and situation of the collateral branches, and upon the force of the circulating current. The direction in which the coagulation principally extends, whether in the arteries or veins, is consequently backwards, from vessels of smaller to those of larger calibre; the formation of the thrombus continuing until it meets with a current sufficiently strong to restore the circulation, which in many cases is as far as the entrance of the next large collateral vessel. The end of the thrombus next the heart is rounded and conical in shape.

The thrombus when once formed either becomes *organized* or *softens*. The former is most frequent in the arteries, the latter in the veins and heart.

Organization.—This consists in the gradual transformation of the thrombus into connective tissue. A thrombus which is undergoing a process of organization gradually diminishes in size, it becomes more and more decolorized, firmer and more fibrous in consistence, its union with the wall of the vessel becomes more intimate, and ultimately it becomes converted into a fibro-cellular cord. These changes are principally owing to the white blood-corpuscles.

Soon after the formation of the thrombus the number of white blood-corpuscles which it contains becomes greatly increased, whilst that of the red ones diminishes. In a somewhat more advanced stage the red corpuscles almost entirely disappear, the fibrin becomes firm and homogeneous, and in addition to the round white corpuscles, numerous anastomosing spindle-shaped cells with oval nuclei make their appearance in the clot. (Fig. 56.) The intercellular material then becomes fibrillated, the walls of the vessel become infiltrated with cells, and numerous new bloodvessels are formed which intersect the thrombus in all directions. These vessels communicate with the cavity of the thrombosed vessel, and with its vasa vasorum. (Fig. 57.) The vascular fibrillated structure into which the thrombus has become organized, gradually undergoes a process of atrophy and contraction, the new vessels disappear, and ultimately it is con-

FIG. 56.

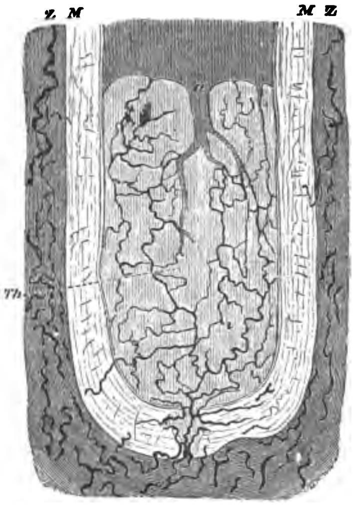


Section of an arterial thrombus thirty-seven days old. *a.* New bloodvessels. *b.* White blood-corpuscles and anastomosing cells. (Rindfleisch.)

verted into a fibro-cellular cord. In some cases it becomes calcified, and thus forms a phlebolith.

Respecting the source from which the large number of new cells which make their appearance in the thrombus are derived, it appears to be most probable that they originate from white blood-corpuscles, and not from cells belonging to the walls of the vessel. Whether, however, they are all of them the offspring of the corpuscles which originally existed in the clot, or whether some of them may not be derived from other sources and subsequently penetrate it, is unknown. From these corpuscles the elongated connective tissue-cells are produced. The exact source from which the fibrillated intercellular material is derived—whether the fibrin itself fibrillates, or whether it disappears, and the fibres are derived from the protoplasm of the cells—is equally uncertain.

FIG. 57.



Longitudinal section of the ligatured end of the crural artery of a dog, fifty days after the application of the ligature. Showing the newly-formed vessels in the thrombus and their communication with the vasa vasorum. *Th.* Thrombus. *M.* Muscular coat. *Z.* External coat and vasa vasorum. x 20. (O. Weber.)

Softening.—If the thrombus does not become organized, it usually undergoes a process of softening and liquefaction: this is most common in the veins and heart. It is probable also that in some cases the thrombus may become absorbed. The softening most frequently commences in the centre of the clot, and gradually extends towards the circumference. The thrombus breaks down into a soft pulpy material, which sometimes has the appearance of pounded cooked meat; and in other cases is distinctly puriform in character. Under the microscope it is seen to consist of albuminous granules, molecular fat, and more or less altered red and white blood-corpuscles. These changes in the clot are frequently owing simply to the disintegration of the fibrin, but in some cases it is probable that the thrombus may suppurate, and that owing to the proliferation of the white blood-corpuscles which it contains it becomes converted into true pus. The whole of the thrombus may thus become softened, or the process may be limited to the more central portions, whilst the external layers become organized. Very frequently as the older portions of the clot are becoming disintegrated and softened, fresh coagulation takes place at its extremities.

CAUSES.—The causes of thrombosis are of two kinds—those which lead to a retardation of the blood-flow, and those in which there is some abnormal condition of the walls of the vessels or of the blood.

1. *Thrombosis from Retardation of the Blood-flow* may result from—

α. *Interruption or narrowing of the vessel.*—This occurs after the application of a ligature. Coagulation commences at the point of contact, and extends as far as the first large collateral branches, thus permanently closing the vessel. The pressure exercised by tumors, cicatricial tissue, extravasations of blood, and the closure of a vessel by the impaction of an embolus, may in the same way by impeding or arresting the circulation cause thrombosis. General obstruction in the capillaries of a part also causes coagulation in the adjacent veins.

β. *Solution of the continuity of the vessel.*—The formation of a thrombus after the division or tearing of a vessel, constitutes the means by which the hemorrhage is immediately arrested: there must either be thrombosis or continuous hemorrhage. In the arteries, the severed end of the vessel contracts and retracts within its sheath, coagulation commences around it, and extends upwards as far as the first large collateral branch. In the veins, hemorrhage is frequently arrested by the valves, and the formation of a thrombus will evidently depend upon the relative situations of the valves and collateral vessels. The hemorrhage from the uterus after the separation of the placenta, is arrested either by uterine contraction or by thrombosis.

γ. *Dilatation of the vessels, or of the heart.*—The most familiar example of thrombosis from this cause, is that which occurs in an aneurism. The greater the amount of dilatation the greater is the retardation of the blood-flow. The coagulation commences at the sides of the vessel, and may extend until it completely fills the cavity. Coagulation from the same cause is not uncommon in the dilated plexuses of the prostate gland. In the heart thrombosis is most frequent in the auricles. It usually commences in the auricular appendix, where there is very little propulsive power, and it may gradually extend into the auricular cavity. It is also met with in the ventricles, commonly commencing here between the columnæ carneæ.

δ. *Diminished cardiac power.*—This is a common cause of thrombosis in the veins. The coagulation commences just behind the flaps of the valves, from which it gradually extends into the cavity of the vessel. This appears to be owing to the force of the current not being sufficiently strong to completely open the valves, and the blood consequently stagnates and coagulates behind them. The crural and iliac veins, the venous plexuses of the back, and the cerebral sinuses, are the situations in which thrombosis from this cause is most frequently met with. It occurs in the course of many chronic exhausting diseases in which the cardiac power becomes diminished, and has long been known under the name of "phlebitis." It is especially frequent in phthisis, cancer, and in chronic diseases of the bones and joints. The state of the blood, which often contains an excess of fibrin, together with the quiescent condition of the patient, materially aid in causing the coagulation.

2. *Thrombosis from Abnormal Conditions of the Vessels or of the Blood.*

a. *Causes in the vessels.*—Any abnormal condition of the walls of a vessel may be a cause of thrombosis. When the wall becomes abnormal it acts as a foreign body, and the blood coagulates upon it, and may continue to do so until the cavity of the vessel becomes filled with coagulum. Thrombi produced in this way are consequently stratified. The walls of a vessel may become altered as the result of inflammatory processes, and inflammation was formerly regarded as the main, if not the only, cause of thrombosis; hence thrombosis in veins is frequently termed "phlebitis" at the present day. Inflammation of veins is certainly rare as a primary condition, although it not unfrequently results from the formation of a thrombus. When occurring primarily, inflammatory processes, both in the arteries and the veins, have their seat in the external and middle coats or in the deeper layers of the intima. They never commence in the lining membrane of the vessel: this only becomes affected secondarily. The vitality of the lining membrane becomes impaired as the result of the inflammatory process, and when this has occurred it acts as a foreign body, and thus there is a tendency for the blood to coagulate upon its surface. In other cases the lining membrane is completely destroyed, and the subjacent diseased tissues thus come into contact with the circulating blood, and in the same way cause the formation of a thrombus. Such inflammatory changes occur in the arteries, constituting the condition known as "atheroma," which, in the smaller vessels, may be a cause of thrombosis. In the heart they constitute endocarditis; and here also, as has been seen, coagulation may take place upon the abnormal surface of the inflammatory vegetations. (See "Endocarditis.")

The walls of a vessel may also become altered and thus thrombosis result, from inflammation or gangrene of the tissues in which it is situated. The vitality of the vessel becomes destroyed and the blood coagulates within it; and by this means the occurrence of hemorrhage is frequently prevented. The projection of new formations, as cancer, into the cavity of vessels, causes in the same way the formation of a thrombus.

β. *Causes in the blood.*—The greater the proportion of fibrin in the blood, the more readily will coagulation take place; hence all those conditions in which the fibrin is increased favor the occurrence of thrombosis. An excess of fibrin, however, is probably never sufficient in itself to determine the formation of a thrombus; it can hence only be regarded as a predisposing cause. It is especially in those conditions in which the circulation is impeded from diminished cardiac power, that an excess of fibrin in the blood becomes an important agent in producing thrombosis.

RESULTS.—The results of thrombosis comprise certain changes in the walls of the vessels, more or less obstruction to the circulation, and embolism. These must be considered separately.

1. *Changes in the vessels.*—More or less alteration in the wall of the vessel is an invariable consequence of the formation of a thrombus. When the thrombus undergoes a process of organization, it becomes, as already described, intimately united with the vascular wall. The latter in the first place becomes infiltrated with cells and considerably thickened, but ultimately, together with the thrombus, gradually atrophies. It is when the thrombus softens and becomes disentegrated that the most important changes take place in the vessel. These changes are of an acute inflammatory nature, and appear to result from the irritating influence of the softened thrombus. They are most frequently observed in the veins, where softening is most liable to occur.

The walls of a vein within which a thrombus is softening are considerably thickened, so that the vessel more resembles an artery. The inner surface has lost its translucency, and is of a dead opaque color. The vasa vasorum are hyperæmic. Under the microscope, the cells of the intima and of the middle and external coats, are found to be considerably increased in number, and numerous white blood-corpuscles are seen infiltrating the different textures. In some cases small collections of pus are seen in the external coat. Similar changes are observed in the arteries.

2. *Obstruction to the circulation.*—The consequences of the obstruction to the circulation which results from the formation of a thrombus, will depend upon the rapidity of its formation, the nature and size of the vessel obstructed, the situation and number of the collateral branches, and the force of the circulating current. When a thrombus forms in a vein of small size and there are numerous collateral vessels, as in the prostatic or uterine plexuses, the circulation is but little interfered with, and no symptoms of obstruction result. If, however, the main trunk of a large vein becomes obliterated, as that of the femoral or iliac veins, the obstruction is followed by hyperæmia, the extent and duration of which will depend upon the facility with which the circulation can be restored by the collateral vessels. Thrombosis in the above-named veins frequently occurs in the latter stages of many chronic diseases, especially in phthisis;

also in the puerperal state, where it gives rise to the condition known as *phlegmasia dolens.* The formation of a thrombus here is followed by œdema and swelling of the limb, which becomes tense, elastic, and painful. In the early stage there may be some cyanosis, but this is usually quickly followed by a pallid whiteness of the surface. There is often more or less tenderness in the course of the vein, which feels enlarged, hard, and knotted, owing to the secondary inflammatory changes in its walls. At the same time there is frequently swelling and tenderness of the lymphatics, which may be seen as red lines traversing the limb. Diffuse inflammation of the skin and subcutaneous cellular tissue may also occur. These changes are owing partly to the mechanical impediment to the circulation, and partly to the obstruction of lymphatics, and to the secondary inflammatory processes in the vein and tissues which ensue. The circulation is usually ultimately restored; but if the impediment has been of long duration, the tissues become thickened, and the limb is left in a hard, indurated, and somewhat enlarged condition.

The formation of a thrombus in an artery is followed in the first place by anæmia of the parts supplied by it; the ultimate result will depend upon the facility with which the circulation can be restored by the collateral vessels. If the circulation is quickly re-established, as is usually the case, the vitality of the tissues may not become impaired; but if not, the part may undergo a process of molecular disintegration and softening, the softened tissue often being surrounded by a zone of hyperæmia which results from the attempt to establish a collateral circulation.

3. *Embolism.*—Portions of the thrombus may be carried away by the circulation, thus constituting embolism. This, which is the most important result of thrombosis, will be considered in the following chapter.

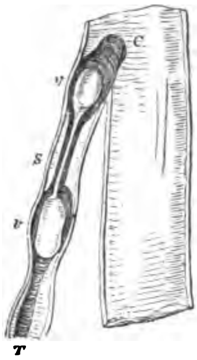
CHAPTER XXXIX.

EMBOLISM.

EMBOLISM is the arrest of solid substances circulating in the blood in vessels which are too small to allow them to pass. The solid substances are termed *emboli.* These are very various in their nature.

By far the most frequent source of emboli are thrombi, portions of which are carried from the seat of their formation by the circulation, and become arrested in distant vessels—thus constituting embolism. A thrombus may give rise to emboli in various ways. It may soften and break down, and if the lumen of the vessel be thus restored, its fragments become distributed by the blood-current. In those cases in which the thrombus does not fill the vessel, portions of it may readily be carried away by the blood passing over it. Perhaps, however, the most frequent way in which a thrombus gives rise to embolism, is by its conical end being broken off by the current of blood from a collateral vessel. The formation of a thrombus, as already described, usually ceases opposite the entrance of a large collateral vessel, and if its conical end project a little way into the cavity of this vessel it may be readily broken off by the blood-current. (Fig. 58.) It is especially venous thrombi which gives rise to embolism: the veins of the leg, the iliac, hypogastric, and jugular veins being amongst the most common sources. Emboli from cardiac thrombi are also exceedingly common; whilst those from arterial are the least frequent.

FIG. 58.



A thrombus in saphenous vein. Showing the projection of the conical end of the thrombus into the femoral vessel. *S* Saphenous vein. *T.* Thrombus. *C* Conical end projecting into femoral vein. At *v v*, opposite the valves, the thrombus is softened. (Virchow.)

Emboli may, however, originate independently of thrombi:

vegetations, and calcareous or atheromatous masses separated from the valves of the heart, or from the inner surface of arteries; portions of new growths, as carcinoma, which having perforated the vessels, have been carried away by the current; parasites which have made their way into the interior of vessels; pigment granules, and other substances, may all constitute emboli.

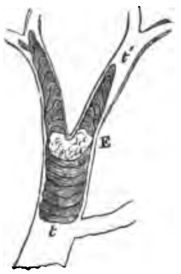
The emboli become arrested in the first vessels they meet with which are too small to allow them to pass: the size of the vessel will consequently depend upon the size of the embolus. They are often so minute that they pass into and become impacted in the smallest capillaries. The seat of impaction is usually at the bifurcation of the vessel, or where, from the giving off of branches, the calibre is diminishing rapidly. (See Fig. 59.) Thus emboli originating in the systemic veins or in the right cardiac cavities, will most commonly become arrested in the vessels of the lungs; those originating in the arteries, the left cardiac cavities, or the pulmonary veins—in the systemic arteries and capillaries, especially in those of the spleen, kidneys, and brain: and those originating in the portal venous system—in the hepatic branches of the portal vein. In some cases, however, the smallest emboli may pass through the capillaries of the lungs and become arrested in those of the kidneys, spleen, or other organs. Thus, with the exception of emboli originating in the portal vessels, the seat of arrest is the arteries or capillaries.

The emboli are usually carried in the direction of the main current; hence those carried by the aortic stream more commonly pass into the thoracic aorta than into the carotid and subclavian vessels, and into the left carotid and renal artery than into the corresponding arteries of the opposite side. Gravitation also influences the direction in which they are carried, especially those of large size which move somewhat more slowly than the blood-stream. Owing to this, they are more common in the lower lobes and posterior parts of the lungs than in the superior and anterior portions of these organs.

The embolus when arrested, may either completely or only partially fill the cavity of the vessel. If, as is frequently the case, the arrest takes place at a point of bifurcation, the embolus may partially fill both branches, allowing a small stream of blood to pass. This may break off portions of it, and so cause secondary emboli, which become impacted in more distant vessels. The amount of obstruction which immediately follows the arrest, will partly depend upon the nature of the embolus itself. If this is from a soft recently-formed thrombus, it will adapt itself to the cavity of the vessel, and so completely occlude it. If, on the other hand, it is irregular in shape and firm in consistence, as a calcified cardiac vegetation, it may not fill the vessel, but allow a small current of blood to pass it.

The arrest of the embolus and the consequent obstruction to the circulation, are followed by the formation of thrombi behind and in front of it, which extend as far as the entrance of the first large collateral vessels. (Fig. 59.) If the embolus does not completely fill the vessel, fibrin is deposited in successive layers upon its surface until the occlusion of the vessel is complete, and then the secondary thrombus extends as in the former case, until it meets with a current of blood strong enough to arrest its progress. If the embolus is a portion of a soft thrombus, it will in most cases be impossible to distinguish it from the secondary thrombus which surrounds it. If, however, it is a calcareous mass, or a portion of an old thrombus, it may usually be distinguished from the more recent secondary coagulum. The changes in these secondary thrombi are similar to those already described as occurring in the primary,—comprising adhesion to the wall of the vessel, softening, and organization.

FIG. 59.



Embolus impacted at the bifurcation of a branch of the pulmonary artery. Showing the formation of thrombi behind and in front of it, and the extension of these as far as the entrance of the next collateral vessels. *E.* Embolus. *t t.* Thrombi. (Virchow.)

RESULTS.—The results of embolism are of two kinds: those depending upon the mechanical obstruction to the circulation, and those produced by the irritating or infective properties of the emboli themselves.

The first series of changes are those occurring in the walls of the vessel within which the embolus becomes arrested. If the embolus possesses no infective properties, being derived from a source where no putrefactive changes are taking place, it, together with the thrombus which it causes to form around and beyond it, simply becomes organized or reabsorbed, and the walls of the vessel become more or less thickened. If, on the other hand, the embolus is impregnated with pus or putrid substances, it is very liable to cause inflammation and sloughing of the walls of the vessel within which it is impacted.

The most important changes, however, resulting from embolism are those which take place in the organ or tissue, the vessels of which have become plugged by the emboli. The first effect of the plugging of a vessel by an embolus, is the arrest of the circulation through it, and if the vessel be the main nutrient or functional artery, this is followed by the sudden cessation of the function and nutrition of the part. Thus, plugging of one of the larger arteries in the brain is followed by sudden loss of consciousness and paralysis (apoplexy); plugging of the pulmonary artery, by sudden asphyxia; and of the coronary arteries, by sudden paralysis of the heart. The subsequent changes will depend upon the structure of the organ, the arrangement of its vessels, and the facility with which a collateral circulation can be established. If the circulation be quickly re-established by the collateral vessels, the part recovers itself without undergoing any structural-change, and its nutrition and functions are restored. If, however, this is not the case, and the nutrient supply is materially interfered with, either by the obstruction of the main vessel, of the principal branches, or of the capillaries themselves—the death of the part must necessarily ensue. The interference with the circulation is in great measure dependent upon the thrombosis which occurs around the impacted embolus. The whole of the tissue thus cut off may suddenly die, or, as is more commonly the case, it undergoes a process of gradual molecular disintegration. This molecular death is in most cases attended by softening, and the softened tissues are usuallly surrounded by a zone of intense hyperæmia, which results from the stress which is thrown upon the collateral vessels. This zone of hyperæmia is very characteristic, and indicates at once the nature of the lesion.

The hyperæmia of the adjacent vessels, which results from the obstruction of the main or of several of the smaller branches, very frequently leads to the extravasation of blood. The distended capillaries give way, and the blood escapes and infiltrates the surrounding tissues. The tract of tissue within which the circulation has thus become arrested from the impaction of the embolus and the resulting thrombosis, and which is more or less extensively infiltrated with blood, is known as a *hemorrhagic infarct.* These hemorrhagic infarcts are very frequently met with, especially in the lungs, spleen, and kidneys. They are red masses of consolidation, and owing to the distribution of the bloodvessels, are usually wedge-shaped, the apex of the cone being towards the centre of the organ. If the circulation is not quickly restored, a process of molecular disentegration and softening commences in the central portions of the infarct. (Fig. 60.) The more complete the obstruction, the more vascular the tissue, and the less the vessels are supported, the greater is the amount of infarction, and the more rapid the softening and disintegration that ensues.

FIG. 60.

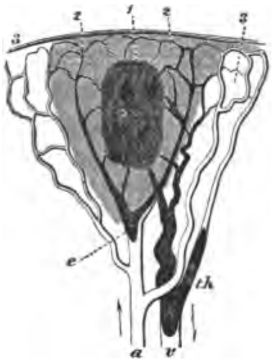


Diagram of an embolic infarct. *a.* Artery obliterated by an embolus (*e*). *v.* Vein filled with a secondary thrombus (*th*). 1. Centre of infarct which is becoming disintegrated. 2. Area of extravasation. 3. Area of collateral hyperæmia. (O. Weber.)

The subsequent changes which take place in the red infarct depend upon its size, upon the extent to which the circulation in it is interfered with, and upon the nature of the embolus which caused the infarction. If the infarct is small and the circulation still continues in parts of it, and if the embolus possesses no infective properties, the coagulated blood may gradually become decolorized, and the mass become partially absorbed or organized. The infarct then changes from a dark red to a brown or yellow tint, the fibrin becomes organized into connective tissue, and the whole gradually contracts, until ultimately a cicatrix may be all that remains to indicate the change. If, however, the infarction is considerable, and the circulation is completely arrested, a process of molecular disintegration and softening commences in the central portions of the tissue which may ultimately involve the whole mass, and the infarct then becomes converted into a pulpy granular material, which may subsequently dry up and become encapsuled. Lastly, if the embolus possesses infective properties, as when it is derived from a part where putrefactive inflammatory changes are going on, it sets up inflammatory processes both in the vessel within which it becomes impacted, and also in the surrounding thrombosed tissue. In this case the infarct rapidly becomes disintegrated, and is converted into a purulent liquid. This is the *embolic abscess*. In all these secondary changes which take place in the infarct, its most external portions are surrounded by a red zone of hyperæmic tissue. This is exceedingly characteristic.

EMBOLISM IN THE BRAIN.

The impaction of emboli within the vessels of the brain is one of the causes of *cerebral softening*. The softening resulting from embolism is, for the most part, entirely dependent upon the obstruction to the circulation caused by the embolus. It is rapidly induced, and is usually attended by the extravasation of blood, and it constitutes by far the most frequent condition known as *acute red softening*. If, however, the interference with the circulation is slight, there may be no extravasation of blood and the process of disintegration may be more gradual, so that the softened portions are white in color, and the condition resembles the chronic white softening already described as resulting from degeneration of the cerebral bloodvessels (see "Fatty Degeneration of the Brain"). The softened tissue will also be white in color when one of the large vessels is obstructed, so that a large portion of one hemisphere loses its vitality. In such cases, only the pia mater and the most superficial layers of the cerebral substance may be hyperæmic. In almost all cases in which softening of the cerebral substance results from embolism, the embolus is arrested in one of the vessels beyond the circle of Willis, because here the circulation cannot be readily restored by the collateral vessels.

When the interference with the circulation is attended by considerable extravasation of blood, the softened portion, in the early stage, is either of a uniform dark red color, or presents numerous red hemorrhagic points. The softening is most marked in the centre, whilst the hyperæmia and redness may extend for some distance around it, so as even to involve the membranes. Under the microscope, the softened portion is seen to consist of broken-down nerve-fibres, altered blood-corpuscles, granules of fat, and the large granular corpuscles which result from the fatty degeneration of the nerve-cells and of the cells of the neuroglia (see Fig. 3, *b*). The surrounding capillaries are dilated and filled with coagula, and the granular corpuscles envelop their walls. In a more advanced stage all trace of nerve structure is lost, the softened mass becomes decolorized, and passes from a dark red color to a chocolate, brown, yellow, or even white. It may liquefy and form a cyst; more commonly, however, it gradually dries up, and a process of repair takes place by the growth of the surrounding neuroglia, which forms a fibrous network in the place of the softened tissue. This contracts, and ultimately a cicatrix with hæmatoidin crystals may be all that remains.

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